

00639253 CAB Accession Number: 780844386

The development of some species and strains of **Eimeria** from single sporozoites and **sporocysts**.

Shirley, M. W.

Houghton Poultry Res. Sta., Houghton, Huntingdon, UK.

Conference Title: No. 17).

Journal of Protozoology vol. 24 (4): p.43A-44A

Publication Year: 1977

ISSN: 0022-3921 --

Language: English

Document Type: Abstract only

Single **sporocysts** of **Eimeria** mivati and **E. acervulina** respectively, were able to establish infections in 7 and 40% of chickens. Single sporozoites were not infective, however.

7/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10099536 BIOSIS NO.: 199598554454

The effect of in ovo oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge.

AUTHOR: Watkins K L(a); Brooks M A; Jeffers T K; Phelps P V; Ricks C A

AUTHOR ADDRESS: (a)Elanco Animal Health, Lilly Corporate Center, Building 13/4 Drop 2047, Indianapolis, IN 46285\*\*USA

JOURNAL: Poultry Science 74 (10):p1597-1602 1995

ISSN: 0032-5791

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: A trial was conducted to investigate the effects of in ovo **Eimeria** maxima inoculation on response to subsequent posthatch challenge with E. maxima. The in ovo treatments were arranged in a 4 x 2 factorial with four in ovo inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop incubation with 50,000 sporulated E. maxima oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in ovo administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in ovo were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in ovo may complete their life-cycle within the developing chick, this experiment provided no evidence that in ovo administration of either E. maxima oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of E. maxima oocysts provided significant protection against subsequent coccidial challenge.

Logging in to Dialog

Trying 31060000009999...Open

DIALOG INFORMATION SERVICES

PLEASE LOGON:

\*\*\*\*\*

ENTER PASSWORD:

\*\*\*\*\*

Welcome to DIALOG

Dialog level 02.05.06D

Last logoff: 12jun02 10:47:46

Logon file405 15jun02 15:04:58

\*\*\* ANNOUNCEMENT \*\*\*

\*\*\*

--Important Notice for Japanese KMKNET Users  
KMKNET will be terminated on 5/31/02. Please  
switch to DLGNET. Please refer to the G-Search  
home page at <http://www.g-search.or.jp>  
for more information.

\*\*\*

--SourceOne patents are now delivered to your  
email inbox as PDF replacing TIFF delivery.  
See HELP SOURCE1 for more information.

\*\*\*

--Important news for public and academic  
libraries. See HELP LIBRARY for more information.

\*\*\*

--Important Notice to Freelance Authors--  
See HELP FREELANCE for more information

\*\*\*

For information about the access to file 43 please see Help News43.

\*\*\*

NEW FILES RELEASED

\*\*\*AGROProjects (File 235)

\*\*\*ARCHIVES OF DERMATOLOGY - SUBSCRIBERS (File 787)

\*\*\*ARCHIVES OF GENERAL PSYCHIATRY -SUBSCRIBERS (File 794)

\*\*\*ARCHIVES OF INTERNAL MEDICINE - SUBSCRIBERS (File 795)

\*\*\*ARCHIVES OF NEUROLOGY - SUBSCRIBERS (File 796)

\*\*\*ARCHIVES OF OPHTHALMOLOGY - SUBSCRIBERS (File 797)

\*\*\*ARCHIVES OF OTOLARYNGOLOGY - SUBSCRIBERS (File 798)

\*\*\*ARCHIVES OF PEDIATRIC & ADOLESCENT MEDICINE-  
Subscribers (File 789)

\*\*\*ARCHIVES OF SURGERY - SUBSCRIBERS (File 800)

\*\*\*JAMA - Journal of the American Medical Association -  
Subscribers (File 785)

\*\*\*MIRA (File 81)

\*\*\*TRADEMARKSCAN-Japan (File 669)

\*\*\*

UPDATING RESUMED

\*\*\*Delphes European Business (File 481)

\*\*\*

RELOADED

\*\*\*CLAIMS/US PATENTS (Files 340, 341, 942)

\*\*\*Kompass Western Europe (590)

\*\*\*D&B - Dun's Market Identifiers (516)

REMOVED

\*\*\*Baton Rouge Advocate (File 382)

\*\*\*Washington Post (File 146)

\*\*\*Books in Print (File 470)

\*\*\*Court Filings (File 793)

\*\*\*Microcomputer Software Guide Online (File 278)  
\*\*\*Publishers, Distributors & Wholesalers of the U.S. (File 450)  
\*\*\*State Tax Today (File 791)  
\*\*\*Tax Notes Today (File 790)  
\*\*\*Worldwide Tax Daily (File 792)

\*\*\*New document supplier\*\*\*

IMED has been changed to INFOTRIE (see HELP OINFOTRI)

>>>Get immediate news with Dialog's First Release news service. First Release updates major newswire databases within 15 minutes of transmission over the wire. First Release provides full Dialog searchability and full-text features. To search First Release files in OneSearch simply BEGIN FIRST for coverage from Dialog's broad spectrum of news wires.

>>> Enter BEGIN HOMEBASE for Dialog Announcements <<<  
>>> of new databases, price changes, etc. <<<  
\*\*\*\*\*

SYSTEM:HOME

Cost is in DialUnits

Menu System II: D2 version 1.7.8 term=ASCII

\*\*\* DIALOG HOMEBASE(SM) Main Menu \*\*\*

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

(c) 2000 The Dialog Corporation plc All rights reserved.

/H = Help

/L = Logoff

/NOMENU = Command Mode

Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).

? dialog

>>Invalid Option Number

\*\*\* DIALOG HOMEBASE(SM) Main Menu \*\*\*

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

(c) 2000 The Dialog Corporation plc All rights reserved.

/H = Help

/L = Logoff

/NOMENU = Command Mode



Enter an option number to view information or to connect to an online service. Enter a BEGIN command plus a file number to search a database (e.g., B1 for ERIC).  
? b 410

15jun02 15:05:00 User226352 Session D636.1  
\$0.00 0.161 DialUnits FileHomeBase  
\$0.00 Estimated cost FileHomeBase  
\$0.00 Estimated cost this search  
\$0.00 Estimated total session cost 0.161 DialUnits

File 410:Chronolog(R) 1981-2002/May  
(c) 2002 The Dialog Corporation

Set Items Description  
--- --

? set hi ;set hi  
HIGHLIGHT set on as ''  
HIGHLIGHT set on as ''  
? b agri

15jun02 15:05:37 User226352 Session D636.2  
\$0.00 0.072 DialUnits File410  
\$0.00 Estimated cost File410  
\$0.13 TELNET  
\$0.13 Estimated cost this search  
\$0.13 Estimated total session cost 0.233 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 5:Biosis Previews(R) 1969-2002/Jun W2  
(c) 2002 BIOSIS  
File 6:NTIS 1964-2002/Jun W5  
(c) 2002 NTIS, Intl Cpyrght All Rights Res  
\*File 6: See HELP CODES6 for a short list of the Subject Heading Codes (SC=, SH=) used in NTIS.  
File 10:AGRICOLA 70-2002/Jun  
(c) format only 2002 The Dialog Corporation  
File 28:Oceanic Abst. 1964-2002/Jun  
(c) 2002 Cambridge Scientific Abstracts  
File 34:SciSearch(R) Cited Ref Sci 1990-2002/Jun W3  
(c) 2002 Inst for Sci Info  
File 44:Aquatic Sci&Fish Abs 1978-2002/May  
(c) 2002 FAO (for ASFA Adv Brd)  
File 50:CAB Abstracts 1972-2002/May  
(c) 2002 CAB International  
\*File 50: Truncating CC codes is recommended for full retrieval.  
See Help News50 for details.  
File 65:Inside Conferences 1993-2002/Jun W2  
(c) 2002 BLDSC all rts. reserv.  
File 76:Life Sciences Collection 1982-2002/Jun  
(c) 2002 Cambridge Sci Abs  
File 94:JICST-EPlus 1985-2002/Apr W3  
(c)2002 Japan Science and Tech Corp(JST)  
\*File 94: There is no data missing. UDs have been adjusted to reflect the current months data. See Help News94 for details.  
File 98:General Sci Abs/Full-Text 1984-2002/May  
(c) 2002 The HW Wilson Co.  
File 99:Wilson Appl. Sci & Tech Abs 1983-2002/May  
(c) 2002 The HW Wilson Co.  
File 117:Water Resour.Abs. 1967-2002/May  
(c) 2002 Cambridge Scientific Abs.  
File 143:Biol. & Agric. Index 1983-2002/May  
(c) 2002 The HW Wilson Co  
File 144:Pascal 1973-2002/Jun W2  
(c) 2002 INIST/CNRS  
File 203:AGRIS 1974-2002/Mar

Dist by NAL, Intl Copr. All rights reserved  
 File 235:AGROProjects 1990-2002/Q3  
 (c) 2002 PJB Publications,Ltd.  
 File 266:FEDRIP 2002/Apr  
 Comp & dist by NTIS, Intl Copyright All Rights Res  
 File 306:Pesticide Fact File 1998/Jun  
 (c) 1998 BCPC  
 \*File 306: File has been updated & reloaded. See HELP NEWS 306. New  
 Bluesheet available in F415 & at URL <http://library.dialog.com/bluesheets>.  
 File 357:Derwent Biotech Res. 1982-2002/Mar W5  
 (c) 2002 Thomson Derwent & ISI  
 \*File 357: Price changes as of 1/1/02. Please see HELP RATES 357.  
 Derwent announces file enhancements. Please see HELP NEWS 357.  
 File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec  
 (c) 1998 Inst for Sci Info

Set Items Description

? s sporocyst? and (Eimeria or tenella or necatrix or acervulina or parecox or  
 brunetti or mitis)

	7320	SPOROCYST?
	24010	EIMERIA
	9761	TENELLA
	2412	NECATRIX
	3412	ACERVULINA
	0	PARECOX
	1198	BRUNETTI
	4783	MITIS
S1	1290	SPOROCYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)

? rd s1

>>>Duplicate detection is not supported for File 235.

>>>Duplicate detection is not supported for File 306.

>>>Records from unsupported files will be retained in the RD set.

...examined 50 records (50)  
 ...examined 50 records (100)  
 ...examined 50 records (150)  
 ...examined 50 records (200)  
 ...examined 50 records (250)  
 ...examined 50 records (300)  
 ...examined 50 records (350)  
 ...examined 50 records (400)  
 ...examined 50 records (450)  
 ...examined 50 records (500)  
 ...examined 50 records (550)  
 ...examined 50 records (600)  
 ...examined 50 records (650)  
 ...examined 50 records (700)  
 ...examined 50 records (750)  
 ...examined 50 records (800)  
 ...examined 50 records (850)  
 ...examined 50 records (900)  
 ...examined 50 records (950)  
 ...examined 50 records (1000)  
 ...examined 50 records (1050)  
 ...examined 50 records (1100)  
 ...examined 50 records (1150)  
 ...examined 50 records (1200)  
 ...examined 50 records (1250)

>>>Record 266:262532 ignored; incomplete bibliographic data, not retained -  
 in RD set

...completed examining records

S2 707 RD S1 (unique items)

? ds

```

Set      Items  Description
S1       1290   SPORO CYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI-
              NA OR PARECOX OR BRUNETTI OR MITIS)
S2       707    RD S1 (unique items)
? t s2 and (immuniz?)
>>>'AND' not allowed in command
? s s2 and (immuniz?)
              707    S2
              246099 IMMUNIZ?
S3       13     S2 AND (IMMUNIZ?)
? t s3/7/all
>>>Format 7 is not valid in file 143

```

```

3/7/1      (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

```

```

07015693   BIOSIS NO.: 000089107577
SELECTION AND CHARACTERIZATION OF A PRECOCIOUS LINE OF EIMERIA
-INTESTINALIS AN INTTESTINAL RABBIT COCCIDIUM
AUTHOR: LICOIS D; COUDERT P; BOIVIN M; DROUET-VIARD F; PROVOT F
AUTHOR ADDRESS: I.N.R.A., LAB. DE PATHOL. DU LAPIN, CENT. DE RECHERCHES DE
TOURS-NOUZILY, 37380 MONNAIE, FR.
JOURNAL: PARASITOL RES 76 (3). 1990. 192-198. 1990
FULL JOURNAL NAME: Parasitology Research
CODEN: PARPE
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

```

ABSTRACT: A precocious line of **Eimeria** intestinalis was obtained by selection for early development of oocysts in rabbits and after six consecutive passages in animals. this line (EiP) was derived from a wild strain (EiO) isolated in 1975 from the caecal content of rabbit with caccidiosis. The prepatent period of the EiP strain was reduced from 215 h to < 144 h, the result being that the oocyst sporulation time was the same for both lines. The excreted and unsporulated oocysts had exactly the same shape, but microscopical examination of the sporulated oocysts showed a marked difference between EiP and EiO strains. A huge refractile globule was located in each of two **sporocysts** of the precocious line, whereas no refractile globule was seen in the other two. The EiP line had a reproductive potential much lower (1000 times) than that of its parent strain EiO and, as judged by the weight gain, mortality and lesions that also occurred in the jejunum and above all in the ileum, its pathogenicity was substantially reduced.

```

3/7/2      (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2002 BIOSIS. All rts. reserv.

```

```

06159456   BIOSIS NO.: 000085122608
ANTIBODY DEVELOPMENT AND CELLULAR IMMUNE RESPONSES IN SHEEP IMMUNIZED
AND CHALLENGED WITH SARCOCYSTIS-TENELLA SPORO CYSTS
AUTHOR: O'DONOGHUE P J; WILKINSON R G
AUTHOR ADDRESS: CENT. VET. LAB., DEP. AGRIC., FROME ROAD, ADELAIDE 5000,
SOUTH AUSTRALIA.
JOURNAL: VET PARASITOL 27 (3-4). 1988. 251-266. 1988
FULL JOURNAL NAME: Veterinary Parasitology
CODEN: VPARD
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

```

ABSTRACT: Four specific-pathogen-free (SPF) sheep were experimentally infected with 103 or 104 Sarcocystis **tenella** (syn. S. ovicanis)

**sporocysts** and another two sheep served as uninfected controls. All sheep were challenged 49 days later by infection with 2.5 times 10<sup>5</sup> **sporocysts** and their humoral and cellular responses to infection and challenge were assessed weekly by enzyme immunoassays and lymphocyte transformation assays. The control sheep died from acute sarcocystosis 29-30 days after challenge, whereas the **immunized** sheep survived and were protected against acute disease. Specific IgM and IgG antibodies were detected in the **immunized** sheep from 28 days after infection onwards. Lymphocytes collected before and after challenge did not exhibit any significant differences in their responses to stimulation with *S. tenella* cystozoite or sporozoite antigens. Furthermore, lymphocytes collected before challenge did not differ from the controls in their responses to stimulation with the mitogens lipopolysaccharide or phytohaemagglutinin. However, lymphocytes collected after challenge did exhibit increased blastogenic responses to stimulation with both mitogens from 21-28 days after challenge onwards. The infected sheep were necropsied 46 days after challenge, and histological and ultrastructural studies revealed numerous infiltrates of lymphocytes, histiocytes and plasma cells in the skeletal muscles, sometimes in association with degenerating parasitic cysts and macrophage myophagia. Parasites were not completely eliminated nor prevented from further establishment, therefore the protective immunity was not sterile but rather a state of premunition.

3/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

05183726 BIOSIS NO.: 000082024347  
ULTRASTRUCTURAL LOCALIZATION OF IMMUNOGLOBULIN A AND IMMUNOGLOBULIN G  
RECEPTORS ON OOCYSTS **SPOROCYSTS** SPOROZOITES AND MEROZOITES OF  
**EIMERIA-FALCIFORMIS**  
AUTHOR: WHITMIRE W M; SPEER C A  
AUTHOR ADDRESS: VETERINARY RESEARCH LAB., MONTANA STATE UNIV., BOZEMAN, MT,  
USA 59717.  
JOURNAL: CAN J ZOOL 64 (3). 1986. 778-784. 1986  
FULL JOURNAL NAME: Canadian Journal of Zoology  
CODEN: CJZOA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The localization of parasite-specific IgA and IgG immunoglobulins on *Eimeria falciformis* oocysts, **sporocysts**, sporozoites, and merozoites was examined by immunoelectron microscopy. Parasites were fixed in glutaraldehyde, incubated with heat-inactivated sera or gut contents from normal or specifically **immunized** mice, reacted with ferritin-conjugated or colloidal gold-conjugated sheep or goat antimouse IgA or IgA antibody and prepared for transmission electron microscopy. Other purified samples of sporozoites or merozoites were exposed to sera or gut contents, fixed in 0.15% glutaraldehyde, and then incubated with ferritin-conjugated or colloidal gold-conjugated sheep or goat antimouse antibody. Parasite-specific IgA and IgG receptors were detected on the plasmalemma of sporozoites and merozoites. Specific IgG receptors were also present on the inner and outer layers of the oocyt wall, and on the inner surface of the **sporocyst** wall. Live sporozoites and merozoites shed immune complexes at their posterior ends. No internal alternations were detected ultrastructurally in sporozoites or merozoites treated with parasite-specific IgA or IgG antibodies.

3/7/4 (Item 4 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04739701 BIOSIS NO.: 000080042828  
EFFECTS OF INTESTINAL CONTENTS FROM NORMAL AND **IMMUNIZED** MICE ON  
SPOROZOITES OF **EIMERIA-FALCIFORMIS**  
AUTHOR: DOUGLASS T G; SPEER C A  
AUTHOR ADDRESS: DEP. MICROBIOL., PUBLIC HEALTH, MICH. STATE UNIV., EAST  
LANSING, MICH.  
JOURNAL: J PROTOZOOLOGY 32 (1). 1985. 156-163. 1985  
FULL JOURNAL NAME: Journal of Protozoology  
CODEN: JPROA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The interaction of *E. falciformis* sporozoites with the intestinal epithelium and with the intestinal contents from the cecum and colon of normal and specifically **immunized** mice was studied by light (LM) and scanning electron (SEM) microscopy. Fecal (FM) and enterocyte-associated (EAM) mucus were removed from the cecum and colon of normal mice and mice that had been **immunized** 1, 6, 12, or 20 days earlier with a series of oral inoculations of *E. falciformis* oocysts. Sporozoite-specific IgA, but neither IgM nor IgG, was detected by the immunofluorescent antibody test in FM and EAM from **immunized** mice. No sporozoite-specific immunoglobulin was detected in normal mice. When examined by LM, sporozoites exposed to all FM and EAM preparations exhibited greater motility and excystation from **sporocysts**. At 4 h after incubation in FM or EAM from normal or immune mice, approx. 10% of the sporozoites appeared damaged, being nonmotile and nonrefractile. Immune FM and EAM caused agglutination of sporozoites and **sporocysts** and oocysts walls of *E. falciformis*, but did not agglutinate those of *E. ferrisi*. Scanning electron microscopy of in vitro interactions between *E. falciformis* sporozoites and intestinal contents revealed that sporozoites exposed to immune EAM were coated with particulate material whereas those exposed to normal EAM were relatively clean. Sporozoites exposed to immune FM were usually embedded within the mucus whereas those exposed to normal FM were situated on top of the mucus. No significant differences occurred between the length/width (L/W) ratios of sporozoites incubated in normal FM and EAM or in PBS. Sporozoites exposed to immune FM had significantly greater L/W ratios than those exposed to normal FM whereas those exposed to immune EAM had significantly shorter L/W ratios than ones exposed to normal EAM. Few of the sporozoites observed on the luminal surface of the colon and cecum of normal mice were covered by mucus and none were altered in shape or showed pellicular damage. Only a few sporozoites were observed on the luminal surface of the colon and cecum of **immunized** mice. Most of these were covered by mucus and some exhibited pellicular alterations.

3/7/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

02196331 BIOSIS NO.: 000064038850  
DEMONSTRATION OF CIRCULATING ANTIBODIES TO **EIMERIA-TENELLA** BY  
THE INDIRECT IMMUNO FLUORESCENT ANTIBODY TEST USING SPOROZOITES AND 2ND  
STAGE SCHIZONTS AS ANTIGEN  
AUTHOR: KOUWENHOVEN B; KUIL H  
JOURNAL: VET PARASITOL 2 (3). 1976 (RECD 1977) 283-292. 1976  
FULL JOURNAL NAME: Veterinary Parasitology  
CODEN: VPARD  
RECORD TYPE: Abstract

ABSTRACT: In the indirect immunofluorescent antibody (IFA) test using sporozoites as an antigen, sera from chickens **immunized** via the natural route were positive in dilutions as high as 1:2048. Serum from a rabbit, **immunized** only to sporozoites by s.c. injection, was positive in a dilution of 1:4096. Non-**immunized** chicken and rabbit

sera were positive in dilutions varying from 1:20-1:64. Sporozoites within **sporocysts** were not stained. In frozen sections of infected chorioallantoic membranes and ceca, sporozoites were not traced with the IFA test with **immunized** chicken or rabbit sera. With the rabbit serum specific diffuse intraepithelial and subepithelial fluorescence was observed in the ceca from 4-11 h after infection. Fluorescence was never associated with the 1st-stage schizonts and gametes, but 2nd-stage schizonts were positive with chicken and rabbit serum. The titers obtained with this antigen were about the same as those obtained with sporozoite smears. The possible presence of common antigens in sporozoites and 2nd stage schizonts is discussed.

3/7/6 (Item 1 from file: 10)  
DIALOG(R)File 10:AGRICOLA  
(c) format only 2002 The Dialog Corporation. All rts. reserv.

2252656 83009940 Holding Library: PAR  
Immunity to sarcocystosis: Modification of intestinal coccidiosis, and disappearance of sarcocysts in dairy goats  
Dubey, J.P.; VPARD  
Amsterdam : , Elsevier Scientific.  
Veterinary parasitology. v. 13 (1) , Aug 1983 p. 23-34.  
ISSN: 0304-4017  
NAL: SF810.V4  
Language: English  
Subfile: PAR; OTHER FOREIGN;  
Document Type: ARTICLE  
Sarcocystis capracanis, dairy goats vaccinated orally with **sporocysts**, development of subclinical sarcocystosis, deaths from intestinal coccidiosis (mixed **Eimeria** species) and Pasturella hemolytica pneumonia, disappearance of sarcocysts in muscles, protection against challenge with lethal doses of S. capracanis

3/7/7 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

02095724 Genuine Article#: KA596 Number of References: 26  
Title: DEVELOPMENT AND CHARACTERIZATION OF MONOCLONAL-ANTIBODIES TO 1ST-GENERATION MEROZOITES OF **EIMERIA**-BOVIS  
Author(s): HAEHER PJ; LINDSAY DS; BLAGBURN BL  
Corporate Source: AUBURN UNIV,COLL VET MED,DEPT PATHOBIOL/AUBURN//AL/36849;  
AUBURN UNIV,COLL VET MED,DEPT PATHOBIOL/AUBURN//AL/36849  
Journal: VETERINARY PARASITOLOGY, 1992, V44, N3-4 (OCT), P321-327  
ISSN: 0304-4017  
Language: ENGLISH Document Type: NOTE  
Abstract: Merozoites of **Eimeria** bovis were harvested from bovine monocyte cell cultures and used to **immunize** BALB/C mice. Spleens from **immunized** mice were removed and the cells fused with mouse myeloma cells. Supernates from resulting hybridoma cell lines were examined for antibodies to first-generation E. bovis merozoites using an indirect immunofluorescent antibody (IFA) assay. Three positive cell lines were identified and cloned by limiting dilution. All three cell lines produced immunoglobulins of the IgG1 isotype that recognized antigens in the anterior half to two-thirds of the merozoites. Specificity of the monoclonal antibodies was examined with the IFA assay against sporozoites of E. bovis, sporozoites and merozoites of **Eimeria** papillata from mice and **Eimeria** tenella from chickens, sporozoites of Isospora suis from pigs, and tachyzoites of Toxoplasma gondii and Neospora caninum from cell cultures. Monoclonal antibodies from the three clones reacted with the anterior end of E. bovis sporozoites, but did not react with the other parasites examined. None of the monoclonal antibodies reacted with merozoite antigens in

immunoblots.

3/7/8 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01739579 CAB Accession Number: 862279080  
Protection against a lethal dose of *Sarcocystis tenella*  
**sporocysts** by pre-exposure of sheep to a sub-lethal dose.  
Cole, D. J. W.; Jonas, W. E.  
New Zealand Journal of Zoology vol. 12 (3): p.443  
Publication Year: 1985  
ISSN: 0301-4223 --  
Language: English  
Document Type: Abstract only

3/7/9 (Item 2 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01034483 CAB Accession Number: 800882822  
Sheep sarcosporidiosis: controlling sarco. Interruption of transmission  
by farm predators and experimental induction of immunity to *Sarcocystis*  
**tenella** in lambs.  
Lightowlers, M. W.; Ford, G. E.  
Vet. Parasit. Group Lab., Inst. of Med. & Vet. Sci., Frome Rd.,  
Adelaide, South Australia.  
Conference Title: Australian Society for Parasitology: Programme and  
abstracts of papers presented at the 24th Conference of the Society, held  
at Flinders University, South Australia, 19-21 May 1980.  
p.(10)  
Publication Year: 1980  
Publisher: -- ., Australia  
Language: English  
Document Type: Miscellaneous  
*Sarcocystis gigantea* cystozoites, irradiated *S. tenella*  
**sporocysts** or *Brucella abortus* lipopolysaccharide in Freund's  
Complete Adjuvant were used as vaccine.

3/7/10 (Item 3 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00579548 CAB Accession Number: 772502563  
Immunity to coccidiosis: interactions in vitro between *Eimeria*  
**tenella** and chicken phagocytic cells.  
Rose, M. E.; Long, P. L.  
Houghton Poultry Res. Sta., Houghton, Huntingdon, Cambs., UK.  
Biochemistry of parasites and host-parasite relationships.  
p.449-455  
Publication Year: 1976  
Editors: Van den Bossche, H.  
Publisher: -- Amsterdam, The: North-Holland Publishing Company.,  
Netherlands  
Language: English  
Document Type: Miscellaneous  
Phagocytic cells (polymorphonuclear leucocytes and macrophages), from  
chickens immunized with *Eimeria tenella* adhere to and  
phagocytose **sporocysts** and sporozoites of the homologous organism  
more actively than do cells from susceptible chickens. To determine  
whether this increased activity of the host cells has any significance in  
protective immunity, the viability of sporozoites incubated in peritoneal

exudate cells from normal and **immunized** chickens was estimated. Sporozoites were inoculated into developing chicken embryos and the infections induced were measured by mortality and haemorrhage, or by oocyst production. No differences were found, suggesting that, although phagocytic cells from specifically **immunized** chickens interact more readily with sporozoites of *E. tenella*, they are not, per se, capable of affecting subsequent development of the parasite. These results, together with those from work in progress, suggest that additional factors, either antibodies and/or the products of the interactions of sensitized lymphocytes with antigens, may be necessary for the full expression of immunity. (AS).

3/7/11 (Item 4 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00106729 CAB Accession Number: 732208844

Biochemical investigation with regard to infection and immunity of **Eimeria acervulina** in the fowl.

Horst, C. J. G. van der; Kouwenhoven, B.  
Clinic Vet. Obstetrics, Yalelaan 7, Utrecht, Netherlands.  
Zeitschrift fur Parasitenkunde vol. 42 (Heft 1): p.23-38  
Publication Year: 1973 --

Language: English

Document Type: Journal article

Biochemical investigation of the fluid surrounding the **sporocysts** in the oocysts ('oocyst fluid') showed the presence of the common amino acids, beta-isoaminobutyric acid, glycerol, an unidentified carbohydrate and proteins. Incubation experiments with labelled glucose revealed the presence of enzymes able to convert glucose into lactic acid and other acids. Inside the **sporocysts** the common amino acids, glycerol and the unidentified carbohydrate were also present, but beta-isoaminobutyric acid did not occur. The carbohydrate binding protein 'carboglutelin', carbohydrate phosphate and small amounts of glucose and fructose were mainly found inside the **sporocysts**. Incubation experiments of intestinal pieces both from **immunized** and non-**immunized** birds with oocyst fluid, **sporocysts** and labelled glucose showed that a stronger reaction took place in **immunized** birds than in those not **immunized** ones. Similar experiments were performed with non-**immunized** birds at different days after a primary infection. The reaction of the intestinal wall, which seemed quite normal again 19 days after infection, was comparable with that observed in **immunized** birds. It is postulated that in the first part of the intestine of **immunized** birds some compounds are present originating from the first infection. These compounds might enhance the reaction between the oocyst fluid and glucose to such an extent that the pH decreases and epithelial cells are pushed off possibly together with the **sporocysts**. Then leakage of serum proteins might occur.

3/7/12 (Item 5 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00106728 CAB Accession Number: 732208843

Histological observations with respect to the immune mechanism in **Eimeria acervulina** infection in the domestic fowl.

Kouwenhoven, B.; Horst, C. J. G. van der  
Poult. Hlth Serv., Oude Rijkssstraatweg 43, Doorn, Netherlands.  
Zeitschrift fur Parasitenkunde vol. 42 (Heft 1): p.11-21  
Publication Year: 1973 --

Language: English

Document Type: Journal article

A strong repulsion of epithelial tissue into the intestinal lumen



associated with a lowered intestinal pH, thin liquid intestinal contents and leakage of serum protein into the lumen was observed in **immunized** birds, some hours after oral reinfection. The extruded epithelial tissue degenerated and died in the lumen; massive numbers of dying cells could also be observed in tissue sections of the intestinal contents. Most likely the sporozoites are pushed off together with the cells. The repulsion was always associated with a prominent swelling of the muscles in the intestinal villi. In the non-immune birds, however, some cell repulsion and a little swelling of the villus muscles was observed, but only at the tips of the villi. In normal uninfected birds the individual contraction of the villus muscles supports the concept of a physiological repulsion of epithelial cells at the so called 'extrusion zone'. In the **immunized** birds the reaction of the tissue after reinfection could be completely suppressed by cortisone treatment. Incubation experiments of intestinal pieces both from **immunized** and nonimmunized birds with oocyst fluid, **sporocysts** and glucose showed similar reactions; the reaction was much stronger in **immunized** than non-**immunized** birds. These results indicate a local tissue immunity. It is postulated that after a primary infection some compounds stay behind in the intestinal wall and these can enhance the reaction of oocyst fluid and **sporocysts** with glucose to such an extent that much lactic acid is formed, which in turn might affect the intestinal wall, resulting in repulsion of the epithelial tissue.

3/7/13 (Item 1 from file: 357)  
 DIALOG(R)File 357:Derwent Biotech Res.  
 (c) 2002 Thomson Derwent & ISI. All rts. reserv.

0044340 DBA Accession No.: 86-02188 PATENT  
 New antigenic protein derivatives from **Eimeria** spp. - e.g.  
**Eimeria tenella**; useful as vaccine or for monoclonal  
 antibody production  
 PATENT ASSIGNEE: Solvay 1985  
 PATENT NUMBER: EP 164176 PATENT DATE: 851211 WPI ACCESSION NO.: 85-312086  
 (8550)  
 PRIORITY APPLIC. NO.: US 734085 APPLIC. DATE: 850516  
 NATIONAL APPLIC. NO.: EP 85200889 APPLIC. DATE: 850605  
 LANGUAGE: English  
 ABSTRACT: A new purified antigen (I) has a mol.wt. of 25,000 and consists  
 of 2 polypeptides of 17,000 and 8,000 mol.wt. joined by a disulfide  
 bond. **Sporocysts** of **Eimeria tenella** are treated with  
 detergent under non-reducing conditions in the presence of  
 protease-inhibitors, and the solubilized membrane proteins are  
 separated (a) by ion-exchange and hydroxyapatite chromatography or (b)  
 by immune precipitation or affinity chromatography. Alternatively,  
 total genomic DNA is isolated from **E. tenella** oocysts, cleaved  
 with restriction enzymes, and the fragments are inserted into vectors.  
 The vectors are screened to identify clones containing (I)-coding  
 sequences which are isolated and used for (I) production. (I) May be  
 used as a vaccine for conferring immunity in fowl against **E.**  
**tenella** and **Eimeria necatrix** infection. A hybridoma  
 line (ATCC HB8561) produces monoclonal antibody Ptn 7.2A4/4 showing  
 activity against (I) which may be used as a vaccine or for affinity  
 purification of (I). The hybridoma was obtained by fusion of spleen  
 cells from mice **immunized** with **E. tenella** sporozoites with  
 SP2/0M cells. (83pp)

? ds

Set	Items	Description
S1	1290	SPORO CYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	707	RD S1 (unique items)
S3	13	S2 AND (IMMUNIZ?)

? s s1 and (ovo or egg?)

1290 S1  
6114 OVO  
559505 EGG?  
S4 22 S1 AND (OVO OR EGG?)  
? s s4 not s3  
22 S4  
13 S3  
S5 22 S4 NOT S3  
? t s5/7/all  
>>>Format 7 is not valid in file 143

5/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10099536 BIOSIS NO.: 199598554454

The effect of in **ovo** oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge.

AUTHOR: Watkins K L(a); Brooks M A; Jeffers T K; Phelps P V; Ricks C A  
AUTHOR ADDRESS: (a)Elanco Animal Health, Lilly Corporate Center, Building 13/4 Drop 2047, Indianapolis, IN 46285\*\*USA  
JOURNAL: Poultry Science 74 (10):p1597-1602 1995  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: A trial was conducted to investigate the effects of in **ovo** **Eimeria** maxima inoculation on response to subsequent posthatch challenge with E. maxima. The in **ovo** treatments were arranged in a 4 x 2 factorial with four in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop incubation with 50,000 sporulated E. maxima oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in **ovo** administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either E. maxima oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of E. maxima oocysts provided significant protection against subsequent coccidial challenge.

5/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07308161 BIOSIS NO.: 000090088052

**EIMERIA**-VITELLINI NEW-SPECIES APICOMPLEXA EIMERIIDAE FROM THE BRAZILIAN TOUCAN RHAMPHASTOS-VITELLINUS-VITELLINUS LICHTENSTEIN AVES PICIFORMES RHAMPHASTIDAE

AUTHOR: LAINSON R; COSTA A M; SHAW J J  
AUTHOR ADDRESS: SECAO PARASITOL., INST. EVANDRO CHAGAS, FUNDACAO SESP, CAIXA POSTAL 3, 66001 BELEM, PA, BRASIL.  
JOURNAL: MEM INST OSWALDO CRUZ RIO J 85 (2). 1990. 199-202. 1990

FULL JOURNAL NAME: Memorias do Instituto Oswaldo Cruz Rio de Janeiro  
CODEN: MIOCA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: **Eimeria** vitellini n. sp., is described from the faeces of the toucan, Rhamphastos v. vitellinus. Oocysts broadly ellipsoidal to oval (egg-shaped), 22.6 .times. 18.3 (20.0-25.0 .times. 16.3-22.5) .mu.m, shape-index (length/width) 1.2 (1.1-1.4). Oocyst wall a single colourless layer about 0.5 .mu.m thick, becoming thinner at one extremity, at which point the oocyst usually ruptures. No oocyst residuum, but 1 or 2 small polar bodies of about 1.0-1.5 .times. 0.50-1.0 .mu.m. **Sporocysts** ellongated ellipsoid (pear-shaped), 14.3 .times. 7.5 (13.8-15.0 .times. 6.9-7.5) .mu.m, shape-index 1.9 (1.8-2.0), with a thin colourless wall bearing a very delicate Stieda body: a conspicuous sub-Stieda body is present. Sporozoites with anterior and posterior refractile bodies and strongly recurved around a bulky, compact **sporocyst** residuum composed of relatively fine granules and spherules.

5/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

03933565 BIOSIS NO.: 000076019131  
COCCIDIA OF BRAZILIAN EDENTATES **EIMERIA**-CYCLOPEI NEW-SPECIES FROM THE  
SILKY ANTEATER CYCLOPES-DIDACTYLUS AND **EIMERIA**-CHOLOEPI NEW-SPECIES  
FROM THE TWO-TOED SLOTH CHOLOEPUS-DIDACTYLUS  
AUTHOR: LAINSON R; SHAW J J  
AUTHOR ADDRESS: CAIXA POSTAL 3, 66000 BELEM, PARA, BRAZIL.  
JOURNAL: SYST PARASITOL 4 (3). 1982. 269-278. 1982  
FULL JOURNAL NAME: Systematic Parasitology  
CODEN: SYPAD  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: *E. cyclopei* sp. nov. is described from the silky anteater, *Cyclopes didactylus*, from Para State, north Brazil. Undifferentiated oocysts, passed in the feces, complete sporulation in 7 days at 26-28.degree. C. Oocysts are ellipsoidal to sub-spherical, with a mean size of 28.1 .times. 23.6 .mu.m; the wall is .apprx. 1.5 to 2.0 .mu.m thick, apparently with an outer thin, colorless membrane and 2 inner, thicker, striated and yellowish layers. There is no micropyle, oocyst residuum or polar body. The mean measurements of **sporocysts** are 19.0 .times. 9.0 .mu.m, and they are slightly asymmetrical with an elongated pear-shaped, plug-shaped Steida body projecting beyond the end of the **sporocyst**. Sporozoites are as long as or longer than the **sporocysts**; the **sporocyst** residuum is scattered between sporozoites in younger specimens and becomes condensed into rounded mass in older ones. The endogenous stages occur in the epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Uninucleate meront, microgamont and macrogamont precursors are recognizable morphologically. Mature meronts are .apprx. 20.0 .times. 15.7 .mu.m, some producing 12-20 merozoites which are .apprx. 8.7 .times. 2.0 .mu.m, and others 10-26 merozoites which are .apprx. 11.4 .times. 2.0 to 15.0 .times. 3.0 .mu.m. Mature microgamonts which are .apprx. 27.5 .times. 24.1 .mu.m produce from 150-170 microgametes of .apprx. 7.1 .times. 1.0 .mu.m. Microgametes have 2 flagella of unequal length. Mature macrogamonts are .apprx. 28.4 .times. 24.5 .mu.m. **Eimeria** choloepi sp. nov. is recorded from the two-toed sloth, *Choloepus didactylus*, from the same area of Brazil. Undifferentiated oocysts, passed in the feces, complete sporulation in 23 days at 26-28.degree. C. Oocysts with a mean size of 23.0 .times. 20.3 .mu.m have a wall .apprx. 2.0 to 2.5 .mu.m thick which is composed of 2 thick, yellowish and striated outer layers and a delicate, colorless

inner one. There is no micropyle, oocyst residuum or polar granule. Mature **sporocysts** with a mean size of 11.3 .times. 7.7 .mu.m are ellipsoidal to **egg**-shaped and have a poorly developed Steida body. The **sporocyst** residuum is composed of a small number of large globules: the sporozoites are longer than the **sporocyst** and strongly recurved. The endogenous stages occur in epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Dimorphic meronts produce 8-18 merozoites which are either .apprx. 13.0 .times. 2.0 .mu.m or .apprx. 13.0 .times. 3.0 .mu.m. Microgamonts produce 50-80 microgametes of .apprx. 8.0 .times. 1.0 .mu.m. Mature macrogamonts are .apprx. 18.3 .times. 17.9 .mu.m.

5/7/4 (Item 4 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

03019059 BIOSIS NO.: 000070044677  
**EIMERIA**-SAMIAE NEW-SPECIES EIMERIIDAE SPOROZOA FROM THE SNAKE  
PYTHON-RETICULATA AND ITS PATHOGENICITY IN THE INTESTINE  
AUTHOR: ISKANDER A R; TADROS G  
AUTHOR ADDRESS: ANIM. HEALTH RES. INST., DOKKI, CAIRO, EGYPT.  
JOURNAL: ZOOL SOC EGYPT BULL 0 (29). 1979 (1980). 66-71. 1979 1980  
FULL JOURNAL NAME: Zoological Society of Egypt Bulletin  
CODEN: ZSEBA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: *E. samiae* sp. nov. is isolated from the intestine and gall-bladder of the snake *P. reticulata*. It is characterized by spherical oocysts, thick banana-like **sporocysts** and ovoid sporozoites like a hen's **egg**. Endogenous stages found in the intestinal mucosa contain elliptical schizonts, subpherical microgametocytes and kidney-shaped macrogametocytes. It produces enteritis characterized by shortening and thickening of the villi and infiltration of inflammatory cells in the latter.

5/7/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

02203210 BIOSIS NO.: 000064045730  
3 NEW **EIMERIA**-SPP PROTOZOA EIMERIIDAE FROM MUSK OXEN OVIBOS-MOSCHATUS  
WITH RE DESCRIPTIONS OF **EIMERIA**-FAUREI **EIMERIA**-GRANULOSA AND  
**EIMERIA**-OVINA FROM MUSK OXEN AND FROM A ROCKY MOUNTAIN BIGHORN  
SHEEP OVIS-CANADENSIS  
AUTHOR: DUSZYNSKI D W; SAMUEL W M; GRAY D R  
JOURNAL: CAN J ZOOL 55 (6). 1977 990-999. 1977  
FULL JOURNAL NAME: Canadian Journal of Zoology  
CODEN: CJZOA  
RECORD TYPE: Abstract

ABSTRACT: Oocytes of *E. moschati* sp. nov., *E. oomingmakensis* sp. nov., and *E. ovibovis* sp. nov. are described from muskoxen (*O. moschatus*) in Alaska, USA, Canada (Alberta, Northwest Territories, Quebec) and Norway. Oocysts of *E. faurei* (Moussu and Marotel, 1902) Martin, 1909, *E. granulosa* Christensen, 1938 and *E. ovina* Levine and Ivens, 1970 are redescribed from muskoxen and from a Rocky Mountain bighorn sheep, *O. canadensis*, from Montana. Ellipsoid oocysts of *E. moschati* are 17-25 .times. 15-21 (20.5 .times. 17.4) .mu.m with ovoid **sporocysts** 9-12 .times. 5-7 (10.8 .times. 6.1) .mu.m. A micropyle, micropyle cap, multiple polar bodies, Steida bodies and **sporocyst** residua are present. Oocysts of *E. oomingmakensis* are ellipsoid, 38-61 .times. 28-38 (47.5 .times. 33.7) .mu.m with ellipsoid **sporocysts** 18-23 .times.

9-12 (20.4 .times. 10.5) .mu.m. A micropyle, Stieda and substieda bodies, and **sporocyst** residua are present. Ellipsoid oocysts of *E. ovibovis* are 20-25 .times. 16-21 (22.9 .times. 18.8) .mu.m with ellipsoid **sporocysts** 11-15 .times. 5-7 (12.7 .times. 6.0) .mu.m. A micropyle, Stieda bodies, and **sporocyst** residua are present. Similarities between these 6 spp. and all other *Eimeria* spp. from ruminants are discussed.

5/7/6 (Item 1 from file: 10)  
DIALOG(R)File 10:AGRICOLA  
(c) format only 2002 The Dialog Corporation. All rts. reserv.

3481720 20488403 Holding Library: AGL

The effect of in **ovo** oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge

Watkins, K.L. Brooks, M.A.; Jeffers, T.K.

Lilly Corporate Center, Indianapolis, IN.

Champaign, IL : Poultry Science Association, 1921-

Poultry science. Oct 1995. v. 74 (10) p. 1597-1602.

ISSN: 0032-5791 CODEN: POSCAL

DNAL CALL NO: 47.8 Am33P

Language: English

Includes references

Place of Publication: Illinois

Subfile: IND; OTHER US (NOT EXP STN, EXT, USDA; SINCE 12/76);

Document Type: Article

A trial was conducted to investigate the effects of in **ovo** *Eimeria* maxima inoculation on response to subsequent posthatch challenge with *E. maxima*. The in **ovo** treatments were arranged in a 4 X 2 factorial with four in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop intubation with 50,000 sporulated *E. maxima* oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determine and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in **ovo** administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either *E. maxima* oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of *E. maxima* oocysts provided significant protection against subsequent coccidial challenge.

5/7/7 (Item 2 from file: 10)  
DIALOG(R)File 10:AGRICOLA  
(c) format only 2002 The Dialog Corporation. All rts. reserv.

1838518 81746930 Holding Library: AGL; AGL

Investigations of the effect of common trade disinfectants on coccidian oocysts and **sporocysts** (*Eimeria*, *Cystoisospora*, *Toxoplasma* and *Sarcocystis*) as well as on ascaridial eggs in suspension tests.

Untersuchungen uber die Wirksamkeit handelsublicher Desinfektionsmittel auf Kokzidien-Oozysten bzw. Sporozysten (*Eimeria*, *Cystoisospora*, *Toxoplasma* und *Sarcocystis*) sowie auf Spulwurmeier (*Ascaris*, *Toxocara*) im Suspensionsversuch / von Dieter Barutzki. -

Barutzki, Dieter,; 1953-

Munchen , (s.n.) , 1980.  
44 p. : ill. ; 21 cm. --  
Munich. Univesitat. Tierarztliche Fakultat. Inaugural-Dissertation ;  
(1980, no. 51)  
NAL: 41.2 M9222 (1980, no.51)  
English summary ; Includes vita.  
Bibliography: p. 39-44.  
Place of Publication: GERMANY, WEST  
Subfile: USDA (US DEPT. AGR); OTHER FOREIGN;  
Document Type: MONOGRAPH

5/7/8 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

07997622 Genuine Article#: 234TU Number of References: 9  
Title: Interactive classification of porcine **Eimeria** spp. by  
computer-assisted image analysis  
Author(s): Plitt A; Imarom S; Joachim A; Dauschies A (REPRINT)  
Corporate Source: TIERARZTLICHEN HSCH HANNOVER, INST PARASITOL, BUNTEWEG  
17/D-30559 HANNOVER//GERMANY/ (REPRINT); TIERARZTLICHEN HSCH  
HANNOVER, INST PARASITOL/D-30559 HANNOVER//GERMANY/; MAHANAKORN UNIV  
TECHNOL, FAC VET MED/BANGKOK 10530//THAILAND/  
Journal: VETERINARY PARASITOLOGY, 1999, V86, N2 (SEP 30), P105-112  
ISSN: 0304-4017 Publication date: 19990930  
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS  
Language: English Document Type: ARTICLE  
Abstract: Digitalized pictures of oocysts of **Eimeria** scabra, E.  
polita, E. deblickei, E. suis, and E. porci were used to develop a  
program routine that allows semi-automatic interactive classification  
of porcine **Eimeria** spp. with a computer-assisted image analysis  
system. Morphological parameters (shape, color, length and width of  
oocysts and length and width of **sporocysts**) were assayed,  
transformed to color and shape factors and used to calculate  
classification indices (GI, SP). Thresholds were defined for these  
indices to allocate oocysts to groups of species and successively to  
single species. In more than 97% of cases the program routine allocated  
digitalized oocysts (n = 175) to the respective species. E. suis was  
always correctly classified. Interactive classification proved to be  
convenient, fast, precise and largely free of individual bias. (C) 1999  
Elsevier Science B.V. All rights reserved.

5/7/9 (Item 2 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

07518748 Genuine Article#: 175YH Number of References: 24  
Title: Differentiation of porcine **Eimeria** spp. by morphologic  
algorithms  
Author(s): Dauschies A (REPRINT) ; Imarom S; Bollwahn W  
Corporate Source: TIERARZTLICHEN HSCH HANNOVER, INST PARASITOL, BUNTEWEG  
17/D-30559 HANNOVER//GERMANY/ (REPRINT); MAHANAKORN UNIV TECHNOL, FAC  
VET MED/BANGKOK 10530//THAILAND/; TIERARZTLICHEN HSCH HANNOVER, KLIN  
KLEINE KLAUENTIERE/D-30173 HANNOVER//GERMANY/  
Journal: VETERINARY PARASITOLOGY, 1999, V81, N3 (MAR 1), P201-210  
ISSN: 0304-4017 Publication date: 19990301  
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS  
Language: English Document Type: ARTICLE  
Abstract: Oocysts of **Eimeria** spp. were isolated from feces of  
naturally infected sows by conventional flotation. Saturated sodium  
chloride solution was superior to zinc chloride, zinc chloride/sodium  
chloride or sugar solution to isolate oocysts. Seven species, namely  
**Eimeria** scabra, E. polita, E. perminuta, E. deblickei, E. suis,

*E. porci* and *E. spinosa*, were identified. The dimensions of oocysts (n = 4088) and **sporocysts** (n = 3594) were measured with an image analysis system; colour and shape of oocysts were estimated and transformed to numerical values. Of the 4088 oocysts approximately 99% were allocated to the correct species by algorithms calculated on the basis of these values. Rough-walled oocysts (*E. scabra*, *E. polita*, *E. perminuta*, *E. spinosa*) could be distinguished without previous sporulation in most cases (>97%). Smooth-walled oocysts require sporulation for further classification and were accurately allocated to species in at least 93% of cases. (C) 1999 Elsevier Science B.V. All rights reserved.

5/7/10 (Item 3 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

04423929 Genuine Article#: TC372 Number of References: 19  
Title: THE EFFECT OF IN **OVO** OOCYST OR **SPOROCYST** INOCULATION ON  
RESPONSE TO SUBSEQUENT COCCIDIAL CHALLENGE  
Author(s): WATKINS KL; BROOKS MA; JEFFERS TK; PHELPS PV; RICKS CA  
Corporate Source: LILLY CORP CTR,BLDG 13-4 DROP 2047/INDIANAPOLIS//IN/46285  
; LILLY RES LABS/GREENFIELD//IN/46140; EMBREX INC/RES TRIANGLE  
PK//NC/27709  
Journal: POULTRY SCIENCE, 1995, V74, N10 (OCT), P1597-1602  
ISSN: 0032-5791

Language: ENGLISH Document Type: ARTICLE

Abstract: A trial was conducted to investigate the effects of in **ovo** *Eimeria* maxima inoculation on response to subsequent posthatch challenge with *E. maxima*. The in **ovo** treatments were arranged in a 4 x 2 factorial with four in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop intubation with 50,000 sporulated *E. maxima* oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in **ovo** administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either *E. maxima* oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of *E. maxima* oocysts provided significant protection against subsequent coccidial challenge.

5/7/11 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03428922 CAB Accession Number: 970804337  
Endoparasite fauna of the stoat (*Mustela erminea* L.) and the weasel (*Mustela nivalis* L.) in Hesse.  
Original Title: Endoparasitenfauna des Hermelins (*Mustela erminea* L.) und Mauswiesels (*Mustela nivalis* L.) in Hessen.  
Peuser, U.  
Institut für Parasitologie, Justus-Liebig Universität Giessen, Giessen, Germany.

iii + 141 pp.

Publication Year: 1996

Publisher: Justus-Liebig-Universität, Fachbereich Veterinärmedizin,  
Giessen. -- Germany

Language: German Summary Language: english

Document Type: Thesis

A total of 102 stoats (*Mustela erminea*) and 100 weasels (*Mustela nivalis*) trapped in Hesse, Germany, between 1983 and 1991 were examined to investigate which endoparasites they were infected with and the prevalences of these parasites. Some had been deep-frozen prior to examination, but there was no significant difference between these and freshly caught animals with regard to parasites present or their prevalences. Macroscopically visible pathological alterations due to parasites were not recorded in any of the animals. 69 stoats (67.6%) and 61 weasels (61%) were infected by one or more endoparasite species; the spectrum of parasites found was similar in the 2 mustelids. The following were detected by faecal examination: oocysts of *Eimeria mustelae* (20 stoats and 11 weasels; 19.6% and 11%) and *Isospora laidlawi* (2 and 0; 2.0% and 0%); **sporocysts** of *Sarcocystis* spp. (7 and 28; 6.9% and 28.0%); and **eggs** of *Taenia mustelae* (16 and 11; 15.7% and 11.0%), *Strongyloides mustelorum* (37 and 34; 36.3% and 34.0%), *Molineus patens* (13 and 13; 12.7% and 13%), *Capillaria putorii* (8 and 2; 7.8% and 2.0%), *C. mustelorum* (1 and 0; 1.0% and 0%), and a trematode, probably *Euparyphium melis* (2 and 2; 1.96% and 2%). The only significant difference in prevalence between the 2 species was for *Sarcocystis* spp.; it is suggested that this difference is due to a different prey spectrum. Figures for the numbers of oocysts, **sporocysts** or **eggs** found in the faeces, indicating the intensity of infection, are presented. The prevalence figures for the helminth species obtained by necroscopy are in complete agreement with those obtained by coprological investigation; figures for the intensity are also given. This is the first record of *I. laidlawi* from wild mustelids in Europe, and the first German record of *M. patens* from weasels, *C. mustelorum* from stoats and *E. mustelae*, *Sarcocystis* spp., *Strongyloides mustelorum* and *C. putorii* from either species. No helminths were found in the cranial cavities, lungs, liver, kidneys or urinary bladder of any animal. Haematological investigation, and enzymatic digestion of the anal sacs (to look for *Capillaria parvanalis*) and the muscles (to look for *Trichinella spiralis*), did not reveal any parasites. The world literature on the endoparasites of stoats and weasels, particularly those found in Europe, is reviewed. A simple key, to enable identification of common endoparasites of the European stoat and weasel at least to genus level, is presented. 220 ref.

5/7/12 (Item 2 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rights reserved.

03213866 CAB Accession Number: 960802594

The effect of in **ovo** oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge.

Watkins, K. L.; Brooks, M. A.; Jeffers, T. K.; Phelps, P. V.; Ricks, C. A.

Lilly Research Laboratories, Box 708, Greenfield, Indiana 46140, USA.

Poultry Science vol. 74 (10): p.1597-1602

Publication Year: 1995

ISSN: 0032-5791 --

Language: English

Document Type: Journal article

A trial was conducted to investigate the effects of in **ovo** *Eimeria maxima* inoculation on response to subsequent posthatch challenge with *E. maxima*. The in **ovo** treatments were arranged in a 4 x 2 factorial with 4 in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and 2 parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged



group, a naive challenged group, and two posthatch inoculated challenged groups. chicks were challenged by crop intubation with 50 000 sporulated *E. maxima* oocysts 10 days posthatch. On day 8 postchallenge, feed intake was determined and birds were weighted and lesions scored. during the brooding period, oocysts were isolated from the faecal material of chicks receiving in **ovo** administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either *E. maxima* oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of *E. maxima* oocysts provided significant protection against subsequent coccidial challenge.  
19 ref.

5/7/13 (Item 3 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02432621 CAB Accession Number: 910881411

**Eimeria** vitellini n.sp. (Apicomplexa: Eimeriidae) from the Brazilian toucan, *Rhamphastos vitellinus vitellinus* Lichtenstein (Aves: Piciformes: Rhamphastidae).

Lainson, R.; Costa, A. M.; Shaw, J. J.

Secao de Parasitologia, Instituto Evandro Chagas, Fundacao SESP, Caixa Postal 3, 66001 Belem, PA, Brazil.

Memorias do Instituto Oswaldo Cruz vol. 85 (2): p.199-202

Publication Year: 1990 --

Language: English

Document Type: Journal article

*E. vitellini* sp. nov., is described from the faeces of *R. v. vitellinus*. The oocysts are broadly ellipsoidal to oval (**egg**-shaped), 22.6 x 18.3 micro m, shape-index (length/width) 1.2. The oocyst wall is a single colourless layer about 0.5 micro m thick, becoming thinner at one extremity, at which point the oocyst usually ruptures. There is no oocyst residuum, but there are 1 or 2 small polar bodies of about 1.0 - 1.5 x 0.5 - 1.0 micro m. The **sporocysts** are elongated and ellipsoid (pear-shaped), 14.3 x 7.5 micro m, shape-index 1.9, with a thin colourless wall bearing a very delicate Stieda body; a conspicuous sub-Stieda body is also present. The sporozoites have anterior and posterior refractile bodies and are strongly recurved around a bulky, compact **sporocyst** residuum composed of relatively fine granules and spherules. 3 ref.

5/7/14 (Item 4 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02381051 CAB Accession Number: 910870762

An investigation of the potential for spread of *Sarcocystis* spp. and other parasites by feral cats.

Langham, N. P. E.; Charleston, W. A. G.

DSIR Land Resources, Havelock North, New Zealand.

New Zealand Journal of Agricultural Research vol. 33 (3): p.429-435

Publication Year: 1990

ISSN: 0028-8233 --

Language: English

Document Type: Journal article

The potential for spread, by feral cats, of parasites infecting sheep and cattle was investigated on 5 km<sup>2</sup> of New Zealand farmland. Faeces were

collected between June 1984 and August 1986. The diet of the cats consisted primarily of rodents (50% by weight) with scavenged sheep (7%) relatively unimportant. Sarcocystis **sporocysts** were found in 4.8% of 63 faecal samples; given the predominance of rodents in the diet, it is more likely that these derived from species infecting rodents rather than farm animals. Toxoplasma gondii oocysts were not found. The frequency of other parasites found was: Toxocara cati (66.7%), Capillaria erinacei (39.7%), C. aerophila (23.8%), Capillaria spp. (7.9%), other nematode **eggs** (27.0%), **Eimeria** (23.8%), Isospora (15.9%), and unidentified oocysts (44.4%). The home range and movements of feral cats were estimated using radiotelemetry. Female cats using barns and pasture had larger home ranges (1.88-2.79 km<sup>2</sup>) than those living in willows (0.37-1.09 km<sup>2</sup>). Adult males and a dispersing subadult male had larger home ranges (2.76-3.00 km<sup>2</sup>) than females. The density of resident feral cats was maintained at about 3.5/km<sup>2</sup> and seasonally supplemented by young and transient cats. The relative importance of feral and farm cats in the distribution of Sarcocystis and T. gondii on farmland and the possibilities of controlling these vectors are discussed. 32 ref.

5/7/15 (Item 5 from file: 50)  
 DIALOG(R)File 50:CAB Abstracts  
 (c) 2002 CAB International. All rts. reserv.

01374273 CAB Accession Number: 822215402  
 Diagnosis of parasites of indigenous birds of prey and owls.  
 Original Title: Zur Diagnostik heimischer Greifvogel- und Eulenparasiten.  
 Frey, H.; Kutzer, E.  
 Inst. Parasit., Veterinarmed. Univ., Linke Bahngasse 11, A-1030 Vienna, Austria.  
 Praktische Tierarzt vol. 63 (10): p.894...902  
 Publication Year: 1982  
 ISSN: 0032-681X --  
 Language: German  
 Document Type: Journal article  
 For the benefit of the veterinarian in practice, suitable methods of faecal examination for bird parasites are discussed and information aiding recognition of protozoon **sporocysts** and oocysts and of the **eggs** of trematodes, cestodes, strongylids, ascarids, capillariids, spirurids and acanthocephalans of Falconiformes and Strigiformes is given. Brief notes on the biology and the importance in Austria of some of the parasites are included. 15 ref.

5/7/16 (Item 6 from file: 50)  
 DIALOG(R)File 50:CAB Abstracts  
 (c) 2002 CAB International. All rts. reserv.

01315288 CAB Accession Number: 820801483  
 Coccidia of Brazilian edentates: **Eimeria** cyclopei n.sp. from the silky anteater, Cyclopes didactylus (Linn.) and **Eimeria** choloepei n.sp. from the two-toed sloth, Choloepus didactylus (Linn.).  
 Lainson, R.; Shaw, J. J.  
 The Wellcome Parasit. Unit, Section of Parasit., Inst. Evandro Chagas, Fundacao Servicos de Saude Publica, 66.000 Belem, Para, Brazil.  
 Systematic Parasitology vol. 4 (3): p.269-278  
 Publication Year: 1982  
 ISSN: 0165-5752 --  
 Language: English  
 Document Type: Journal article  
**Eimeria** cyclopei n.sp. is described from Cyclopes didactylus from Para State, north Brazil. Undifferentiated oocysts, passed in the faeces, completed sporulation in 7 days at 26 to 28 deg C. Oocysts are ellipsoidal to sub-spherical, 28.1 X 23.6 mu m: the wall is approx equal to 1.5 to 2.0

mu m thick, with an outer thin, colourless membrane and 2 inner, thicker, striated and yellowish layers. There is no micropyle, oocyst residuum or polar body. The **sporocysts** measure 19.0 X 9.0 mu m and are slightly asymmetrical, elongate pear-shape, with a plug-shaped Steida body projecting beyond the end of the **sporocyst**. Sporozoites are as long as or longer than the **sporocysts**. The **sporocyst** residuum is scattered between sporozoites in younger specimens and condensed into a rounded mass in older ones. The endogenous stages occur in the epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Uninucleate meront, microgamont and macrogamont precursors are recognizable morphologically. Mature meronts are approx equal to 20.0 X 15.7 mu m, some produce 12 to 20 merozoites which are approx equal to 8.7 X 2.0 mu m, and others 10 to 26 merozoites which are approx equal to 11.4 X 2.0 to 15.0 X 3.0 mu m. Mature microgamonts, approx equal to 27.5 X 24.1 mu m, produce from 150 to 170 microgametes approx equal to 7.1 X 1.0 mu m; microgametes have 2 flagella of unequal length. Mature macrogamonts are approx equal to 28.4 X 24.5 mu m. **Eimeria chloepi** n.sp. is described from Chloepus didactylus from the same area. Undifferentiated oocysts, passed in the faeces, completed sporulation in 23 days at 26 to 28 deg C. Oocysts 23.0 X 20.3 mu m, have a wall approx equal to 2.0 to 2.5 mu m thick composed of 2 thick, yellowish and striated outer layers and a delicate, colourless inner one. There is no micropyle, oocyst residuum or polar granule. Mature sporocysts, 11.3 X 7.1 mu m, are ellipsoidal to egg-shaped and have a poorly developed Steida body. The **sporocyst** residuum is composed of a small number of large globules. The sporozoites are longer than the **sporocyst** and strongly recurved. The endogenous stages occur in epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Dimorphic meronts produce 8 to 18 merozoites which are either approx equal to 13.0 X 2.0 mu m or approx equal to 13.0 X 3.0 mu m. Microgamonts produce 50 to 80 microgametes of approx equal to 8.0 X 1.0 mu m. Mature macrogamonts are approx equal to 18.3 X 17.9 mu m. (AS). 7 ref.

5/7/17 (Item 7 from file: 50)  
 DIALOG(R)File 50:CAB Abstracts  
 (c) 2002 CAB International. All rts. reserv.

01037388 CAB Accession Number: 810886955

Efficacy of commercial disinfectants against coccidial oocysts and **sporocysts** (**Eimeria**, Cystoisospora, Toxoplasma and Sarcocystis) and against eggs of Ascaris and Toxocara in suspension experiments.

Original Title: Untersuchungen uber die Wirksamkeit handelsublicher Desinfektionsmittel auf Kokzidien-Oozysten bzw. Sporozysten (**Eimeria**, Cystoisospora, Toxoplasma und Sarcocystis) sowie auf Spulwurmeier (Ascaris, Toxocara) im Suspensionsversuch.

Barutzki, D.

44 pp.

Publication Year: 1980

Publisher: Ludwig-Maximilians Universitat, Munchen. --

Language: German Summary Language: english

Document Type: Thesis

Secondary Journal Source: Veterinary Bulletin 51, 4589.

One-day-old unsporulated **E. tenella** oocysts were damaged after 5 min by 6% Dekaseptol, and after 20 min by 5% Lomasept, 5% Incicoc and 5% Lysococ. Increasing the concentrations of active ingredients by 50% decreased the time of action: **E. tenella** oocysts were damaged after 2 min by Dekaseptol and after 5 min by Lomasept, Incicoc and Lysococ. The increased concentrations showed no effect even after 2 h against the other 1-day-old unsporulated oocysts (**E. brunetti**, **C. canis**, Toxoplasma gondii). Sensitivity of the oocysts to the disinfectants increased with their age. The agents reduced the rate of sporogony or even damaged the oocysts. Freshly sporulated oocysts were more resistant. **E. tenella** survived 60 min of contact and **E. brunetti** 90 min of contact with

6% Dekaseptol, 5% Lomasept, 5% Incicoc and 5% Lysococ. Solutions containing 50% more of the active ingredients did not prevent either excystation by *C. canis* or development of cysts in brains of mice by *T. gondii*, even after 20 min. of contact. After storage of the sporulated oocysts, only *C. canis* was damaged after 2 h of contact with 7.5% Incico. Fresh **sporocysts** of *S. suicanis* were damaged by 5% Incicoc after 10 min and the 6- to 8-month old **sporocysts** were also sensitive to 6% Dekaseptol after 60 min. *S. muris* survived 2 h of contact with disinfectants. ADDITIONAL ABSTRACT: Unembryonated and embryonated **eggs** of *A. suum* and *T. canis* were damaged by 5% Incicoc after 10 min., unembryonated *T. canis* **eggs** only after 2 min., whereas the other agents showed inadequate effects. 76 ref.

5/7/18 (Item 8 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01034707 CAB Accession Number: 810883118

Note on mammalian coccidia at Lucknow Zoo.

Agrawal, R. D.; Ahluwalia, S. S.; Bhatia, B. B.; Chauhan, P. P. S.  
Coll. of Vet. Sci. & Anim. Husb., Chandrashekhar Azad Univ. of Agric. & Tech., Mathura, Uttar Pradesh 281 002, India.

Indian Journal of Animal Sciences vol. 51 (1): p.125-128

Publication Year: 1981

ISSN: 0367-8318 --

Language: English

Document Type: Journal article

At Lucknow Zoo, India, 5 new species of *Isospora* and one of **Eimeria** were recovered from the faeces of a variety of mammals. *I. pantheri* n.sp. from *Panthera (Leo) leo* differs from *I. leonina* and *Isospora* sp. (Pande et al. 1970) by having ellipsoidal and larger oocysts (33.1 to 40.3 x 20.2 to 25.9  $\mu$ m). It further differs from *I. leonina* in the absence of polar granules and by having larger sporozoites and from *Isospora* sp. by having ovoid **sporocysts**. *I. mohini* n.sp., also from *P. (L.) leo*, differs from all the above species by having smaller (18.7 to 27.4 x 14.4 to 20.2  $\mu$ m) and ovoid to broadly ellipsoidal oocysts with a length:width ratio 1.18:1.40 and small elongate sporozoites. *I. leopardi* n.sp. from *Neofelis nebulosa* is characterised by **egg**-shaped and large oocysts (38.9 to 44.6 x 23.0 to 27.4  $\mu$ m), the **sporocystic** residuum present as scattered granules and by shape of the **sporocyst** (ovoid). *I. ursi* n.sp. from *Melursus ursinus* differs from *I. fonsecai* (occurring in *Ursus arctos*) by larger oocysts (34.6 to 43.2 x 20.2 to 30.0  $\mu$ m) with a length:width ratio of 1.40 to 1.73. *I. hippopotami* n.sp. from *Hippopotamus amphibius* has ovoid to broadly ellipsoidal oocysts, no micropyle, polar cap, oocystic residuum or polar granules, subspherical to ovoid **sporocyst** residuum present as clumps and 4 elongated pear-shaped sporozoites. **Eimeria** *ailuri* n.sp. from *Ailurus fulgens* has ellipsoidal oocysts, no micropyle, polar cap, oocystic residuum or polar granules, has ellipsoidal **sporocyst** with a small stieda body at the narrower end and **sporocystic** residuum present as an aggregation of dark granules in the centre. Apart from *I. pantheri* and *I. mohini*, all the other new species represent new host records for coccidia. 4 ref.

5/7/19 (Item 9 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00639669 CAB Accession Number: 780847102

**Eimeria** *gundii* n.sp. (Protozoa: Eimeriidae) from Tunisian gundi (*Ctenodactylus gundi*).

Mishra, G. S.; Gonzalez, J. P.  
Pasteur Inst., Tunis, Tunisia.

Annales de Parasitologie Humaine et Comparee vol. 53 (2): p.241-243

Publication Year: 1978

ISSN: 0003-4150 --

Language: English

Document Type: Journal article

Of 17 *Ctenodactylus gundi* caught in southern Tunisia, 11 had coccidial oocysts in their faeces and, at autopsy, in the intestinal mucosa. 20 oocysts measured 20 to 27 by 18 to 23  $\mu$ m (average 23.6 by 20.3  $\mu$ m). The oocyst wall was 1.0 to 1.5  $\mu$ m thick, no micropyle was seen, there was no polar granule and the oocyst residuum measured 8 to 10  $\mu$ m. The **sporocysts** were 9 to 12 by 5 to 7  $\mu$ m (average 10.6 by 6.6  $\mu$ m); there was a stieda body at the narrower end of the **sporocyst** and the sporozoites, which measured 6 to 8 by 2.5 to 3  $\mu$ m, surrounded a small compact granular residuum. This is the first report of an **Eimeria** species in the *gundi*. It is named **Eimeria gundii** n.sp. ADDITIONAL ABSTRACT: In Tunisia, nematode and cestode **eggs** were found in the faeces of all of 17 *Ctenodactylus gundi*. Trichostrongylidae, Trichuridae, Oxyuridae and Hymenolepididae were found in the intestines at post-mortem examination.

5/7/20 (Item 10 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

00639253 CAB Accession Number: 780844386

The development of some species and strains of **Eimeria** from single sporozoites and **sporocysts**.

Shirley, M. W.

Houghton Poultry Res. Sta., Houghton, Huntingdon, UK.

Conference Title: No. 17).

Journal of Protozoology vol. 24 (4): p.43A-44A

Publication Year: 1977

ISSN: 0022-3921 --

Language: English

Document Type: Abstract only

Single **sporocysts** of **Eimeria mivati** and **E. acervulina** respectively, were able to establish infections in 7 and 40% of chickens. Single sporozoites were not infective, however.

5/7/21 (Item 1 from file: 76)

DIALOG(R)File 76:Life Sciences Collection

(c) 2002 Cambridge Sci Abs. All rts. reserv.

01409827 2356841

**Eimeria vitellini** n. sp. (Apicomplexa: Eimeriidae) from the Brazilian toucan, *Rhamphastos vitellinus vitellinus* lichtenstein (Aves: Piciformes: Rhamphastidae).

Lainson, R.; Costa, A.M.; Shaw, J.J.

Secao Parasitol., Inst. Evandro Chagas, Fund. SESP, Caixa Postal 3, 66001 Belem, Brazil

MEM. INST. OSWALDO CRUZ. vol. 85, no. 2, pp. 199-202 (1990.)

DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH

SUBFILE: Microbiology Abstracts Section C: Algology, Mycology and Protozoology

**Eimeria vitellini** n. sp., is described from the faeces of the toucan, *Rhamphastos v. vitellinus*. Oocysts broadly ellipsoidal to oval (**egg-shaped**), shape-index. Oocyst wall a single colourless layer about 0.5  $\mu$ m thick, becoming thinner at one extremity, at which point the oocyst usually ruptures. No oocyst residuum, but 1 or 2 small polar bodies of about 1.0-1.5 x 0.5-1.0  $\mu$ m. **Sporocysts** elongated ellipsoid shape-index 1.9, with a thin colourless wall bearing a very delicate Stieda body: a conspicuous sub-Stieda body is present. Sporozoites with anterior

and posterior refractile bodies and strongly recurved around a bulky, compact **sporocyst** residuum composed of relatively fine granules and spherules.

5/7/22 (Item 1 from file: 203)  
DIALOG(R)File 203:AGRIS  
Dist by NAL, Intl Copr. All rights reserved. All rts. reserv.

00818648 AGRIS No: 737833

Investigations on the efficiency of commercial disinfectants on coccidian oocysts and **sporocysts** (**Eimeria**, *Cystoisospora*, *Toxoplasma* and *Sarcocystis*) and ascaridial **eggs** (*Ascaris*, *Toxocara*) in suspension tests (Untersuchungen ueber die Wirksamkeit handelsueblicher Desinfektionsmittel auf Kokzidien-Oozysten bzw. Sporozysten (**Eimeria**, *Cystoisospora*, *Toxoplasma* und *Sarcocystis*) sowie auf Spulwurmeier (*Ascaris*, *Toxocara*) im Suspensionsversuch)

Barutzki, D.

Muenchen Univ. (Germany, F.R.). Tieraerztliche Fakultaet

Thesis Degree: Inaugural-Diss. (Dr. med. vet.)

Publisher: , Muenchen (Germany, F.R.), 1980, 44 p.

Notes: 13 tables. Bibliography p. 39-44

Language: German Summary Language: German, English

Place of Publication: Germany, F.R.

Availability: Muenchen Univ. (Germany, F.R.). Universitaetsbibliothek

Document Type: Monograph, Dissertation, Bibliography, Summary,

Nonconventional Literature

Journal Announcement: 0803 Record input by CEC (Commission of European Communities)

? ds

Set	Items	Description
S1	1290	SPOROCYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	707	RD S1 (unique items)
S3	13	S2 AND (IMMUNIZ?)
S4	22	S1 AND (OVO OR EGG?)
S5	22	S4 NOT S3

? s s2 and (vaccin?)

707 S2

441920 VACCIN?

S6 6 S2 AND (VACCIN?)

? s s6 not s3

6 S6

13 S3

S7 3 S6 NOT S3

? t s7/7/all

>>>Format 7 is not valid in file 143

7/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10099536 BIOSIS NO.: 199598554454

The effect of in ovo oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge.

AUTHOR: Watkins K L(a); Brooks M A; Jeffers T K; Phelps P V; Ricks C A

AUTHOR ADDRESS: (a)Elanco Animal Health, Lilly Corporate Center, Building 13/4 Drop 2047, Indianapolis, IN 46285\*\*USA

JOURNAL: Poultry Science 74 (10):p1597-1602 1995

ISSN: 0032-5791

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: A trial was conducted to investigate the effects of in ovo **Eimeria** maxima inoculation on response to subsequent posthatch challenge with *E. maxima*. The in ovo treatments were arranged in a 4 x 2 factorial with four in ovo inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop incubation with 50,000 sporulated *E. maxima* oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in ovo administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in ovo were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in ovo may complete their life-cycle within the developing chick, this experiment provided no evidence that in ovo administration of either *E. maxima* oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of *E. maxima* oocysts provided significant protection against subsequent coccidial challenge.

7/7/2 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03362946 CAB Accession Number: 970802085  
Comparison of the genomic fingerprints generated by the random amplification of polymorphic DNA between precocious lines and parental strains of **Eimeria** spp. from the rabbit.  
Cere, N.; Licois, D.; Humbert, J. F.  
INRA, Laboratoire de Pathologie du Lapin, Station de Pathologie Aviaire et de Parasitologie, F-37380 Nouzilly, France.  
Parasitology Research vol. 83 (3): p.300-302  
Publication Year: 1997  
ISSN: 0044-3255 --  
Language: English  
Document Type: Journal article  
Precocious strains of **Eimeria** spp. which are being investigated as possible live **vaccines** for coccidiosis in rabbits are characterized by large refractile bodies in their **sporocysts**, which suggest genetic differences from parental strains. The random amplified polymorphic DNA (RAPD) technique was used to compare the genome of precocious and wild-type strains of *E. intestinalis* and *E. magna* from rabbits. Although 80 primers were used alone and in combination, no differences were detected in the patterns of precocious and parental strains of both species. It is suggested that only a small number of genes is involved in the trait for precociousness. 18 ref.

7/7/3 (Item 1 from file: 357)  
DIALOG(R)File 357:Derwent Biotech Res.  
(c) 2002 Thomson Derwent & ISI. All rts. reserv.

0045342 DBA Accession No.: 86-03190  
Identification and characterization of the gene for a major surface antigen of **Eimeria tenella** - sporozoite and **sporocyst**  
(conference abstract)  
AUTHOR: Files J G; Paul L S; Kuhn I; Gabe J D  
CORPORATE AFFILIATE: Codon  
CORPORATE SOURCE: Codon, 430 Valley Drive, Brisbane, CA 94005, USA.  
JOURNAL: J.Cell.Biochem. (Suppl.10A, 147) 1986

CODEN: 5210J

LANGUAGE: English

ABSTRACT: **Eimeria tenella** is a species of Coccidia that is responsible for severe losses to the poultry industry. A major surface antigen from **E. tenella** sporozoites and **sporocysts** has been identified, purified and sequenced. This protein sequence was used to design oligonucleotide hybridization probes for the gene encoding the antigen. Libraries of **E. tenella** genomic DNA were constructed in phage vectors lambda-gt-wes-lambda-b and lambda-1059. A clone positive to all of the probes was isolated from each library and was sequenced. DNA sequence shows that these clones contain the entire gene encoding the antigen. The protein encoded by this gene has a structure consistent with that of a eukaryotic cell surface protein. (0 ref)

? ds

Set	Items	Description
S1	1290	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	707	RD S1 (unique items)
S3	13	S2 AND (IMMUNIZ?)
S4	22	S1 AND (OVO OR EGG?)
S5	22	S4 NOT S3
S6	6	S2 AND (VACCIN?)
S7	3	S6 NOT S3

? b biochem

15jun02 15:20:16 User226352 Session D636.3  
\$4.32 0.771 DialUnits File5  
\$19.25 11 Type(s) in Format 7  
\$19.25 11 Types  
\$23.57 Estimated cost File5  
\$0.41 0.069 DialUnits File6  
\$0.41 Estimated cost File6  
\$0.55 0.204 DialUnits File10  
\$4.05 3 Type(s) in Format 7  
\$4.05 3 Types  
\$4.60 Estimated cost File10  
\$0.68 0.116 DialUnits File28  
\$0.68 Estimated cost File28  
\$8.73 0.511 DialUnits File34  
\$9.70 2 Type(s) in Format 7  
\$8.40 2 Type(s) in Format 15  
\$18.10 4 Types  
\$26.83 Estimated cost File34  
\$1.16 0.216 DialUnits File44  
\$1.16 Estimated cost File44  
\$3.87 0.859 DialUnits File50  
\$32.00 16 Type(s) in Format 7  
\$32.00 16 Types  
\$35.87 Estimated cost File50  
\$0.31 0.082 DialUnits File65  
\$0.31 Estimated cost File65  
\$2.48 0.485 DialUnits File76  
\$1.85 1 Type(s) in Format 7  
\$1.85 1 Types  
\$4.33 Estimated cost File76  
\$0.43 0.122 DialUnits File94  
\$0.43 Estimated cost File94  
\$0.21 0.086 DialUnits File98  
\$0.21 Estimated cost File98  
\$0.11 0.046 DialUnits File99  
\$0.11 Estimated cost File99  
\$0.28 0.053 DialUnits File117  
\$0.28 Estimated cost File117  
\$0.19 0.078 DialUnits File143  
\$0.19 Estimated cost File143



\$1.21 0.347 DialUnits File144  
 \$1.21 Estimated cost File144  
 \$0.36 0.151 DialUnits File203  
 \$1.45 1 Type(s) in Format 7  
 \$1.45 1 Types  
 \$1.81 Estimated cost File203  
 \$1.14 0.042 DialUnits File235  
 \$1.14 Estimated cost File235  
 \$0.21 0.059 DialUnits File266  
 \$0.21 Estimated cost File266  
 \$0.31 0.036 DialUnits File306  
 \$0.31 Estimated cost File306  
 \$1.79 0.105 DialUnits File357  
 \$5.40 2 Type(s) in Format 7  
 \$5.40 2 Types  
 \$7.19 Estimated cost File357  
 \$1.98 0.116 DialUnits File434  
 \$1.98 Estimated cost File434  
 OneSearch, 21 files, 4.553 DialUnits FileOS  
 \$3.25 TELNET  
 \$116.08 Estimated cost this search  
 \$116.21 Estimated total session cost 4.786 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 5: Biosis Previews(R) 1969-2002/Jun W2  
 (c) 2002 BIOSIS  
 File 6: NTIS 1964-2002/Jun W5  
 (c) 2002 NTIS, Intl Cpyrght All Rights Res  
 \*File 6: See HELP CODES6 for a short list of the Subject Heading Codes  
 (SC=, SH=) used in NTIS.  
 File 34: SciSearch(R) Cited Ref Sci 1990-2002/Jun W3  
 (c) 2002 Inst for Sci Info  
 File 40: Enviroline(R) 1975-2002/May  
 File 41: Pollution Abs 1970-2002/Jul  
 (c) 2002 Cambridge Scientific Abstracts  
 File 50: CAB Abstracts 1972-2002/May  
 (c) 2002 CAB International  
 \*File 50: Truncating CC codes is recommended for full retrieval.  
 See Help News50 for details.  
 File 65: Inside Conferences 1993-2002/Jun W2  
 (c) 2002 BLDSC all rts. reserv.  
 File 68: Env.Bib. 1972-2002/Apr  
 (c) 2002 Internl Academy at Santa Barbara  
 File 71: ELSEVIER BIOBASE 1994-2002/Jun W2  
 (c) 2002 Elsevier Science B.V.  
 File 73: EMBASE 1974-2002/Jun W2  
 (c) 2002 Elsevier Science B.V.  
 \*File 73: For information about Explode feature please  
 see Help News73.  
 File 76: Life Sciences Collection 1982-2002/Jun  
 (c) 2002 Cambridge Sci Abs  
 File 77: Conference Papers Index 1973-2002/May  
 (c) 2002 Cambridge Sci Abs  
 File 94: JICST-EPlus 1985-2002/Apr W3  
 (c) 2002 Japan Science and Tech Corp(JST)  
 \*File 94: There is no data missing. UDs have been adjusted to reflect  
 the current months data. See Help News94 for details.  
 File 98: General Sci Abs/Full-Text 1984-2002/May  
 (c) 2002 The HW Wilson Co.  
 File 103: Energy SciTec 1974-2002/Jun B1  
 (c) 2002 Contains copyrighted material  
 \*File 103: For access restrictions see Help Restrict.  
 File 143: Biol. & Agric. Index 1983-2002/May  
 (c) 2002 The HW Wilson Co  
 File 144: Pascal 1973-2002/Jun W2

(c) 2002 INIST/CNRS  
 File 155:MEDLINE(R) 1966-2002/Jun W2  
 \*File 155: Daily alerts are now available. This file has  
 been reloaded. Accession numbers have changed.  
 File 156:ToxFile 1966-2002/Feb W4  
 (c) 2002  
 File 162:CAB HEALTH 1983-2002/May  
 (c) 2002 CAB INTERNATIONAL  
 \*File 162: Truncating CC codes is recommended for full retrieval.  
 See Help News162 for details.  
 File 172:EMBASE Alert 2002/Jun W2  
 (c) 2002 Elsevier Science B.V.  
 File 305:Analytical Abstracts 1980-2002/May W4  
 (c) 2002 Royal Soc Chemistry  
 \*File 305: Frequency of updates and Alerts changing to weekly.  
 See HELP NEWS 305.  
 File 369:New Scientist 1994-2002/Jun W1  
 (c) 2002 Reed Business Information Ltd.  
 File 370:Science 1996-1999/Jul W3  
 (c) 1999 AAAS  
 \*File 370: This file is closed (no updates). Use File 47 for more current  
 information.  
 File 399:CA SEARCH(R) 1967-2002/UD=13624  
 (c) 2002 AMERICAN CHEMICAL SOCIETY  
 \*File 399: Use is subject to the terms of your user/customer agreement.  
 RANK charge added; see HELP RATES 399.  
 File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec  
 (c) 1998 Inst for Sci Info

Set Items Description

? s sporocyst? and (Eimeria or tenella or necatrix or acervulina or parecox or  
 brunetti or mitis)

	9078	SPOROCYST?
	26120	EIMERIA
	10690	TENELLA
	2421	NECATRIX
	3717	ACERVULINA
	1	PARECOX
	1366	BRUNETTI
	7291	MITIS
S1	1547	SPOROCYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)

? ds

Set	Items	Description
S1	1547	SPOROCYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)

? rd s1

...examined	50 records	(50)
...examined	50 records	(100)
...examined	50 records	(150)
...examined	50 records	(200)
...examined	50 records	(250)
...examined	50 records	(300)
...examined	50 records	(350)
...examined	50 records	(400)
...examined	50 records	(450)
...examined	50 records	(500)
...examined	50 records	(550)
...examined	50 records	(600)
...examined	50 records	(650)
...examined	50 records	(700)
...examined	50 records	(750)
...examined	50 records	(800)

...examined 50 records (850)  
...examined 50 records (900)  
...examined 50 records (950)  
...examined 50 records (1000)  
...examined 50 records (1050)  
...examined 50 records (1100)  
...examined 50 records (1150)  
...examined 50 records (1200)  
...examined 50 records (1250)  
...examined 50 records (1300)  
...examined 50 records (1350)  
...examined 50 records (1400)  
...examined 50 records (1450)  
...examined 50 records (1500)  
...completed examining records

Processing

S2 715 RD S1 (unique items)  
? s s2 and (ovo or egg?)  
715 S2  
9651 OVO  
655710 EGG?  
S3 14 S2 AND (OVO OR EGG?)  
? t s3/7/all  
>>>Format 7 is not valid in file 143

3/7/1 (Item 1 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10099536 BIOSIS NO.: 199598554454

The effect of in **ovo** oocyst or **sporocyst** inoculation on response to subsequent coccidial challenge.

AUTHOR: Watkins K L(a); Brooks M A; Jeffers T K; Phelps P V; Ricks C A  
AUTHOR ADDRESS: (a)Elanco Animal Health, Lilly Corporate Center, Building  
13/4 Drop 2047, Indianapolis, IN 46285\*\*USA  
JOURNAL: Poultry Science 74 (10):p1597-1602 1995  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: A trial was conducted to investigate the effects of in **ovo** **Eimeria** maxima inoculation on response to subsequent posthatch challenge with E. maxima. The in **ovo** treatments were arranged in a 4 x 2 factorial with four in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and **sporocyst**). Four control treatments included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch inoculated challenged groups. Chicks were challenged by crop incubation with 50,000 sporulated E. maxima oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in **ovo** administration of **sporocysts** in the amnion and **sporocysts** or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either E. maxima oocysts or **sporocysts** will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of E. maxima oocysts provided significant protection against subsequent coccidial challenge.

3/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07308161 BIOSIS NO.: 000090088052

**EIMERIA**-VITELLINI NEW-SPECIES APICOMPLEXA EIMERIIDAE FROM THE  
BRAZILIAN TOUCAN RHAMPHASTOS-VITELLINUS-VITELLINUS LICHTENSTEIN AVES  
PICIFORMES RHAMPHASTIDAE

AUTHOR: LAINSON R; COSTA A M; SHAW J J

AUTHOR ADDRESS: SECAO PARASITOL., INST. EVANDRO CHAGAS, FUNDACAO SESP,  
CAIXA POSTAL 3, 66001 BELEM, PA, BRASIL.

JOURNAL: MEM INST OSWALDO CRUZ RIO J 85 (2). 1990. 199-202. 1990

FULL JOURNAL NAME: Memorias do Instituto Oswaldo Cruz Rio de Janeiro

CODEN: MIOCA

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: **Eimeria** vitellini n. sp., is described from the faeces of the toucan, Rhamphastos v. vitellinus. Oocysts broadly ellipsoidal to oval (**egg**-shaped), 22.6 .times. 18.3 (20.0-25.0 .times. 16.3-22.5) .mu.m, shape-index (length/width) 1.2 (1.1-1.4). Oocyst wall a single colourless layer about 0.5 .mu.m thick, becoming thinner at one extremity, at which point the oocyst usually ruptures. No oocyst residuum, but 1 or 2 small polar bodies of about 1.0-1.5 .times. 0.50-1.0 .mu.m. **Sporocysts** ellongated ellipsoid (pear-shaped), 14.3 .times. 7.5 (13.8-15.0 .times. 6.9-7.5) .mu.m, shape-index 1.9 (1.8-2.0), with a thin colourless wall bearing a very delicate Stieda body: a conspicuous sub-Stieda body is present. Sporozoites with anterior and posterior refractile bodies and strongly recurved around a bulky, compact **sporocyst** residuum composed of relatively fine granules and spherules.

3/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

03933565 BIOSIS NO.: 000076019131

COCCIDIA OF BRAZILIAN EDENTATES **EIMERIA**-CYCLOPEI NEW-SPECIES FROM THE  
SILKY ANTEATER CYCLOPES-DIDACTYLUS AND **EIMERIA**-CHOLOEPI NEW-SPECIES  
FROM THE TWO-TOED SLOTH CHOLOEPUS-DIDACTYLUS

AUTHOR: LAINSON R; SHAW J J

AUTHOR ADDRESS: CAIXA POSTAL 3, 66000 BELEM, PARA, BRAZIL.

JOURNAL: SYST PARASITOL 4 (3). 1982. 269-278. 1982

FULL JOURNAL NAME: Systematic Parasitology

CODEN: SYPAD

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: **E. cyclopei** sp. nov. is described from the silky anteater, Cyclopes didactylus, from Para State, north Brazil. Undifferentiated oocysts, passed in the feces, complete sporulation in 7 days at 26-28.degree. C. Oocysts are ellipsoidal to sub-spherical, with a mean size of 28.1 .times. 23.6 .mu.m; the wall is .apprx. 1.5 to 2.0 .mu.m thick, apparently with an outer thin, colorless membrane and 2 inner, thicker, striated and yellowish layers. There is no micropyle, oocyst residuum or polar body. The mean measurements of **sporocysts** are 19.0 .times. 9.0 .mu.m, and they are slightly asymmetrical with an elongated pear-shaped, plug-shaped Steida body projecting beyond the end of the **sporocyst**. Sporozoites are as long as or longer than the **sporocysts**; the **sporocyst** residuum is scattered between sporozoites in younger specimens and becomes condensed into rounded mass in older ones. The endogenous stages occur in the epithelial cells of the

ileum, on the luminal side of the host-cell nucleus. Uninucleate meront, microgamont and macrogamont precursors are recognizable morphologically. Mature meronts are .apprx. 20.0 .times. 15.7 .mu.m, some producing 12-20 merozoites which are .apprx. 8.7 .times. 2.0 .mu.m, and others 10-26 merozoites which are .apprx. 11.4 .times. 2.0 to 15.0 .times. 3.0 .mu.m. Mature microgamonts which are .apprx. 27.5 .times. 24.1 .mu.m produce from 150-170 microgametes of .apprx. 7.1 .times. 1.0 .mu.m. Microgametes have 2 flagella of unequal length. Mature macrogamonts are .apprx. 28.4 .times. 24.5 .mu.m. **Eimeria** choloepi sp. nov. is recorded from the two-toed sloth, Choloepus didactylus, from the same area of Brazil. Undifferentiated oocysts, passed in the feces, complete sporulation in 23 days at 26-28.degree. C. Oocysts with a mean size of 23.0 .times. 20.3 .mu.m have a wall .apprx. 2.0 to 2.5 .mu.m thick which is composed of 2 thick, yellowish and striated outer layers and a delicate, colorless inner one. There is no micropyle, oocyst residuum or polar granule. Mature **sporocysts** with a mean size of 11.3 .times. 7.7 .mu.m are ellipsoidal to **egg**-shaped and have a poorly developed Steida body. The **sporocyst** residuum is composed of a small number of large globules: the sporozoites are longer than the **sporocyst** and strongly recurved. The endogenous stages occur in epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Dimorphic meronts produce 8-18 merozoites which are either .apprx. 13.0 .times. 2.0 .mu.m or .apprx. 13.0 .times. 3.0 .mu.m. Microgamonts produce 50-80 microgametes of .apprx. 8.0 .times. 1.0 .mu.m. Mature macrogamonts are .apprx. 18.3 .times. 17.9 .mu.m.

3/7/4 (Item 4 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

03019059 BIOSIS NO.: 000070044677  
**EIMERIA**-SAMIAE NEW-SPECIES EIMERIIDAE SPOROZOA FROM THE SNAKE  
 PYTHON-RETICULATA AND ITS PATHOGENICITY IN THE INTESTINE  
 AUTHOR: ISKANDER A R; TADROS G  
 AUTHOR ADDRESS: ANIM. HEALTH RES. INST., DOKKI, CAIRO, EGYPT.  
 JOURNAL: ZOOL SOC EGYPT BULL 0 (29). 1979 (1980). 66-71. 1979 1980  
 FULL JOURNAL NAME: Zoological Society of Egypt Bulletin  
 CODEN: ZSEBA  
 RECORD TYPE: Abstract  
 LANGUAGE: ENGLISH

ABSTRACT: *E. samiae* sp. nov. is isolated from the intestine and gall-bladder of the snake *P. reticulata*. It is characterized by spherical oocysts, thick banana-like **sporocysts** and ovoid sporozoites like a hen's **egg**. Endogenous stages found in the intestinal mucosa contain elliptical schizonts, subpherical microgametocytes and kidney-shaped macrogametocytes. It produces enteritis characterized by shortening and thickening of the villi and infiltration of inflammatory cells in the latter.

3/7/5 (Item 5 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

02203210 BIOSIS NO.: 000064045730  
 3 NEW **EIMERIA**-SPP PROTOZOA EIMERIIDAE FROM MUSK OXEN OVIBOS-MOSCHATUS  
 WITH RE DESCRIPTIONS OF **EIMERIA**-FAUREI **EIMERIA**-GRANULOSA AND  
**EIMERIA**-OVINA FROM MUSK OXEN AND FROM A ROCKY MOUNTAIN BIGHORN  
 SHEEP OVIS-CANADENSIS  
 AUTHOR: DUSZYNSKI D W; SAMUEL W M; GRAY D R  
 JOURNAL: CAN J ZOOL 55 (6). 1977 990-999. 1977  
 FULL JOURNAL NAME: Canadian Journal of Zoology  
 CODEN: CJZOA

RECORD TYPE: Abstract

ABSTRACT: Oocytes of *E. moschati* sp. nov., *E. oomingmakensis* sp. nov., and *E. ovibovis* sp. nov. are described from muskoxen (*O. moschatus*) in Alaska, USA, Canada (Alberta, Northwest Territories, Quebec) and Norway. Oocysts of *E. faurei* (Moussu and Marotel, 1902) Martin, 1909, *E. granulosa* Christensen, 1938 and *E. ovina* Levine and Ivens, 1970 are redescribed from muskoxen and from a Rocky Mountain bighorn sheep, *O. canadensis*, from Montana. Ellipsoid oocysts of *E. moschati* are 17-25 .times. 15-21 (20.5 .times. 17.4) .mu.m with ovoid **sporocysts** 9-12 .times. 5-7 (10.8 .times. 6.1) .mu.m. A micropyle, micropyle cap, multiple polar bodies, Stieda bodies and **sporocyst** residua are present. Oocysts of *E. oomingmakensis* are ellipsoid, 38-61 .times. 28-38 (47.5 .times. 33.7) .mu.m with ellipsoid **sporocysts** 18-23 .times. 9-12 (20.4 .times. 10.5) .mu.m. A micropyle, Stieda and substieda bodies, and **sporocyst** residua are present. Ellipsoid oocysts of *E. ovibovis* are 20-25 .times. 16-21 (22.9 .times. 18.8) .mu.m with ellipsoid **sporocysts** 11-15 .times. 5-7 (12.7 .times. 6.0) .mu.m. A micropyle, Stieda bodies, and **sporocyst** residua are present. Similarities between these 6 spp. and all other *Eimeria* spp. from ruminants are discussed.

3/7/6 (Item 1 from file: 50)  
DIALOG(R) File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03428922 CAB Accession Number: 970804337  
Endoparasite fauna of the stoat (*Mustela erminea* L.) and the weasel (*Mustela nivalis* L.) in Hesse.  
Original Title: Endoparasitenfauna des Hermelins (*Mustela erminea* L.) und Mauswiesels (*Mustela nivalis* L.) in Hessen.  
Peuser, U.  
Institut fur Parasitologie, Justus-Liebig Universitat Giessen, Giessen, Germany.  
iii + 141 pp.  
Publication Year: 1996  
Publisher: Justus-Liebig-Universitat, Fachbereich Veterinarmedizin, Giessen. -- Germany  
Language: German Summary Language: english  
Document Type: Thesis  
A total of 102 stoats (*Mustela erminea*) and 100 weasels (*Mustela nivalis*) trapped in Hesse, Germany, between 1983 and 1991 were examined to investigate which endoparasites they were infected with and the prevalences of these parasites. Some had been deep-frozen prior to examination, but there was no significant difference between these and freshly caught animals with regard to parasites present or their prevalences. Macroscopically visible pathological alterations due to parasites were not recorded in any of the animals. 69 stoats (67.6%) and 61 weasels (61%) were infected by one or more endoparasite species; the spectrum of parasites found was similar in the 2 mustelids. The following were detected by faecal examination: oocysts of *Eimeria mustelae* (20 stoats and 11 weasels; 19.6% and 11%) and *Isospora laidlawi* (2 and 0; 2.0% and 0%); **sporocysts** of *Sarcocystis* spp. (7 and 28; 6.9% and 28.0%); and **eggs** of *Taenia mustelae* (16 and 11; 15.7% and 11.0%), *Strongyloides mustelorum* (37 and 34; 36.3% and 34.0%), *Molineus patens* (13 and 13; 12.7% and 13%), *Capillaria putorii* (8 and 2; 7.8% and 2.0%), *C. mustelorum* (1 and 0; 1.0% and 0%), and a trematode, probably *Euparyphium melis* (2 and 2; 1.96% and 2%). The only significant difference in prevalence between the 2 species was for *Sarcocystis* spp.; it is suggested that this difference is due to a different prey spectrum. Figures for the numbers of oocysts, **sporocysts** or **eggs** found in the faeces, indicating the intensity of infection, are presented. The prevalence figures for the helminth species obtained by necroscopy are in complete agreement with those obtained by coprological investigation; figures for

the intensity are also given. This is the first record of *I. laidlawi* from wild mustelids in Europe, and the first German record of *M. patens* from weasels, *C. mustelorum* from stoats and *E. mustelae*, *Sarcocystis* spp., *Strongyloides mustelorum* and *C. putorii* from either species. No helminths were found in the cranial cavities, lungs, liver, kidneys or urinary bladder of any animal. Haematological investigation, and enzymatic digestion of the anal sacs (to look for *Capillaria paranalisis*) and the muscles (to look for *Trichinella spiralis*), did not reveal any parasites. The world literature on the endoparasites of stoats and weasels, particularly those found in Europe, is reviewed. A simple key, to enable identification of common endoparasites of the European stoat and weasel at least to genus level, is presented. 220 ref.

3/7/7 (Item 2 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02432621 CAB Accession Number: 910881411

**Eimeria** vitellini n.sp. (Apicomplexa: Eimeriidae) from the Brazilian toucan, *Rhamphastos vitellinus vitellinus* Lichtenstein (Aves: Piciformes: Rhamphastidae).

Lainson, R.; Costa, A. M.; Shaw, J. J.

Secao de Parasitologia, Instituto Evandro Chagas, Fundacao SESP, Caixa Postal 3, 66001 Belem, PA, Brazil.

Memorias do Instituto Oswaldo Cruz vol. 85 (2): p.199-202

Publication Year: 1990 --

Language: English

Document Type: Journal article

*E. vitellini* sp. nov., is described from the faeces of *R. v. vitellinus*. The oocysts are broadly ellipsoidal to oval (**egg**-shaped), 22.6 x 18.3 micro m, shape-index (length/width) 1.2. The oocyst wall is a single colourless layer about 0.5 micro m thick, becoming thinner at one extremity, at which point the oocyst usually ruptures. There is no oocyst residuum, but there are 1 or 2 small polar bodies of about 1.0 - 1.5 x 0.5 - 1.0 micro m. The **sporocysts** are elongated and ellipsoid (pear-shaped), 14.3 x 7.5 micro m, shape-index 1.9, with a thin colourless wall bearing a very delicate Stieda body; a conspicuous sub-Stieda body is also present. The sporozoites have anterior and posterior refractile bodies and are strongly recurved around a bulky, compact **sporocyst** residuum composed of relatively fine granules and spherules. 3 ref.

3/7/8 (Item 3 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02381051 CAB Accession Number: 910870762

An investigation of the potential for spread of *Sarcocystis* spp. and other parasites by feral cats.

Langham, N. P. E.; Charleston, W. A. G.

DSIR Land Resources, Havelock North, New Zealand.

New Zealand Journal of Agricultural Research vol. 33 (3): p.429-435

Publication Year: 1990

ISSN: 0028-8233 --

Language: English

Document Type: Journal article

The potential for spread, by feral cats, of parasites infecting sheep and cattle was investigated on 5 km<sup>2</sup> of New Zealand farmland. Faeces were collected between June 1984 and August 1986. The diet of the cats consisted primarily of rodents (50% by weight) with scavenged sheep (7%) relatively unimportant. *Sarcocystis* **sporocysts** were found in 4.8% of 63 faecal samples; given the predominance of rodents in the diet, it is more likely that these derived from species infecting rodents rather than farm animals. *Toxoplasma gondii* oocysts were not found. The frequency of

other parasites found was: *Toxocara cati* (66.7%), *Capillaria erinacei* (39.7%), *C. aerophila* (23.8%), *Capillaria* spp. (7.9%), other nematode **eggs** (27.0%), **Eimeria** (23.8%), *Isospora* (15.9%), and unidentified oocysts (44.4%). The home range and movements of feral cats were estimated using radiotelemetry. Female cats using barns and pasture had larger home ranges (1.88-2.79 km<sup>2</sup>) than those living in willows (0.37-1.09 km<sup>2</sup>). Adult males and a dispersing subadult male had larger home ranges (2.76-3.00 km<sup>2</sup>) than females. The density of resident feral cats was maintained at about 3.5/km<sup>2</sup> and seasonally supplemented by young and transient cats. The relative importance of feral and farm cats in the distribution of *Sarcocystis* and *T. gondii* on farmland and the possibilities of controlling these vectors are discussed. 32 ref.

3/7/9 (Item 4 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01374273 CAB Accession Number: 822215402  
Diagnosis of parasites of indigenous birds of prey and owls.  
Original Title: Zur Diagnostik heimischer Greifvogel- und Eulenparasiten.  
Frey, H.; Kutzer, E.  
Inst. Parasit., Veterinarmed. Univ., Linke Bahngasse 11, A-1030 Vienna, Austria.  
Praktische Tierarzt vol. 63 (10): p.894...902  
Publication Year: 1982  
ISSN: 0032-681X --  
Language: German  
Document Type: Journal article  
For the benefit of the veterinarian in practice, suitable methods of faecal examination for bird parasites are discussed and information aiding recognition of protozoon **sporocysts** and oocysts and of the **eggs** of trematodes, cestodes, strongylids, ascarids, capillariids, spirurids and acenthocephalans of Falconiformes and Strigiformes is given. Brief notes on the biology and the importance in Austria of some of the parasites are included. 15 ref.

3/7/10 (Item 5 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01315288 CAB Accession Number: 820801483  
Coccidia of Brazilian edentates: **Eimeria** cyclopei n.sp. from the silky anteater, *Cyclopes didactylus* (Linn.) and **Eimeria** choloepi n.sp. from the two-toed sloth, *Choloepus didactylus* (Linn.).  
Lainson, R.; Shaw, J. J.  
The Wellcome Parasit. Unit, Section of Parasit., Inst. Evandro Chagas, Fundacao Servicos de Saude Publica, 66.000 Belem, Para, Brazil.  
Systematic Parasitology vol. 4 (3): p.269-278  
Publication Year: 1982  
ISSN: 0165-5752 --  
Language: English  
Document Type: Journal article  
**Eimeria** cyclopei n.sp. is described from *Cyclopes didactylus* from Para State, north Brazil. Undifferentiated oocysts, passed in the faeces, completed sporulation in 7 days at 26 to 28 deg C. Oocysts are ellipsoidal to sub-spherical, 28.1 X 23.6  $\mu$ m: the wall is approx equal to 1.5 to 2.0  $\mu$ m thick, with an outer thin, colourless membrane and 2 inner, thicker, striated and yellowish layers. There is no micropyle, oocyst residuum or polar body. The **sporocysts** measure 19.0 X 9.0  $\mu$ m and are slightly asymmetrical, elongate pear-shape, with a plug-shaped Steida body projecting beyond the end of the **sporocyst**. Sporozoites are as long as or longer than the **sporocysts**. The **sporocyst** residuum is



scattered between sporozoites in younger specimens and condensed into a rounded mass in older ones. The endogenous stages occur in the epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Uninucleate meront, microgamont and macrogamont precursors are recognizable morphologically. Mature meronts are approx equal to 20.0 X 15.7  $\mu$  m, some produce 12 to 20 merozoites which are approx equal to 8.7 X 2.0  $\mu$  m, and others 10 to 26 merozoites which are approx equal to 11.4 X 2.0 to 15.0 X 3.0  $\mu$  m. Mature microgamonts, approx equal to 27.5 X 24.1  $\mu$  m, produce from 150 to 170 microgametes approx equal to 7.1 X 1.0  $\mu$  m; microgametes have 2 flagella of unequal length. Mature macrogamonts are approx equal to 28.4 X 24.5  $\mu$  m. **Eimeria choloepi** n.sp. is described from *Choloepus didactylus* from the same area. Undifferentiated oocysts, passed in the faeces, completed sporulation in 23 days at 26 to 28 deg C. Oocysts 23.0 X 20.3  $\mu$  m, have a wall approx equal to 2.0 to 2.5  $\mu$  m thick composed of 2 thick, yellowish and striated outer layers and a delicate, colourless inner one. There is no micropyle, oocyst residuum or polar granule. Mature sporocysts, 11.3 X 7.1  $\mu$  m, are ellipsoidal to egg-shaped and have a poorly developed Steida body. The **sporocyst** residuum is composed of a small number of large globules. The sporozoites are longer than the **sporocyst** and strongly recurved. The endogenous stages occur in epithelial cells of the ileum, on the luminal side of the host-cell nucleus. Dimorphic meronts produce 8 to 18 merozoites which are either approx equal to 13.0 X 2.0  $\mu$  m or approx equal to 13.0 X 3.0  $\mu$  m. Microgamonts produce 50 to 80 microgametes of approx equal to 8.0 X 1.0  $\mu$  m. Mature macrogamonts are approx equal to 18.3 X 17.9  $\mu$  m. (AS). 7 ref.

3/7/11 (Item 6 from file: 50)  
 DIALOG(R) File 50:CAB Abstracts  
 (c) 2002 CAB International. All rts. reserv.

01037388 CAB Accession Number: 810886955  
 Efficacy of commercial disinfectants against coccidial oocysts and **sporocysts** (**Eimeria**, *Cystoisospora*, *Toxoplasma* and *Sarcocystis*) and against **eggs** of *Ascaris* and *Toxocara* in suspension experiments.

Original Title: Untersuchungen uber die Wirksamkeit handelsublicher Desinfektionsmittel auf Kokzidien-Oozysten bzw. Sporozysten (**Eimeria**, *Cystoisospora*, *Toxoplasma* und *Sarcocystis*) sowie auf Spulwurmeier (*Ascaris*, *Toxocara*) im Suspensionsversuch.

Barutzki, D.

44 pp.

Publication Year: 1980

Publisher: Ludwig-Maximilians Universitat, Munchen. --

Language: German Summary Language: english

Document Type: Thesis

Secondary Journal Source: Veterinary Bulletin 51, 4589.

One-day-old unsporulated *E. tenella* oocysts were damaged after 5 min by 6% Dekaseptol, and after 20 min by 5% Lomasept, 5% Incicoc and 5% Lysococ. Increasing the concentrations of active ingredients by 50% decreased the time of action: *E. tenella* oocysts were damaged after 2 min by Dekaseptol and after 5 min by Lomasept, Incicoc and Lysococ. The increased concentrations showed no effect even after 2 h against the other 1-day-old unsporulated oocysts (*E. brunetti*, *C. canis*, *Toxoplasma gondii*). Sensitivity of the oocysts to the disinfectants increased with their age. The agents reduced the rate of sporogony or even damaged the oocysts. Freshly sporulated oocysts were more resistant. *E. tenella* survived 60 min of contact and *E. brunetti* 90 min of contact with 6% Dekaseptol, 5% Lomasept, 5% Incicoc and 5% Lysococ. Solutions containing 50% more of the active ingredients did not prevent either excystation by *C. canis* or development of cysts in brains of mice by *T. gondii*, even after 20 min. of contact. After storage of the sporulated oocysts, only *C. canis* was damaged after 2 h of contact with 7.5% Incico. Fresh **sporocysts** of *S. suicanis* were damaged by 5% Incicoc after 10

min and the 6- to 8-month old **sporocysts** were also sensitive to 6% Dekaseptol after 60 min. *S. muris* survived 2 h of contact with disinfectants. ADDITIONAL ABSTRACT: Unembryonated and embryonated **eggs** of *A.suum* and *T. canis* were damaged by 5% Incicoc after 10 min., unembryonated *T. canis* **eggs** only after 2 min., whereas the other agents showed inadequate effects. 76 ref.

3/7/12 (Item 7 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01034707 CAB Accession Number: 810883118  
Note on mammalian coccidia at Lucknow Zoo.  
Agrawal, R. D.; Ahluwalia, S. S.; Bhatia, B. B.; Chauhan, P. P. S.  
Coll. of Vet. Sci. & Anim. Husb., Chandrashekhar Azad Univ. of Agric. & Tech., Mathura, Uttar Pradesh 281 002, India.  
Indian Journal of Animal Sciences vol. 51 (1): p.125-128  
Publication Year: 1981  
ISSN: 0367-8318 --  
Language: English  
Document Type: Journal article  
At Lucknow Zoo, India, 5 new species of *Isospora* and one of **Eimeria** were recovered from the faeces of a variety of mammals. *I. pantheri* n.sp. from *Panthera (Leo) leo* differs from *I. leonina* and *Isospora* sp. (Pande et al. 1970) by having ellipsoidal and larger oocysts (33.1 to 40.3 x 20.2 to 25.9  $\mu$  m). It further differs from *I. leonina* in the absence of polar granules and by having larger sporozoites and from *Isospora* sp. by having ovoid **sporocysts**. *I. mohini* n.sp., also from *P. (L.) leo*, differs from all the above species by having smaller (18.7 to 27.4 x 14.4 to 20.2  $\mu$  m) and ovoid to broadly ellipsoidal oocysts with a length:width ratio 1.18:1.40 and small elongate sporozoites. *I. leopardi* n.sp. from *Neofelis nebulosa* is characterised by **egg-shaped** and large oocysts (38.9 to 44.6 x 23.0 to 27.4  $\mu$  m), the **sporocystic** residuum present as scattered granules and by shape of the **sporocyst** (ovoid). *I. ursi* n.sp. from *Melursus ursinus* differs from *I. fonsecai* (occurring in *Ursus arctos*) by larger oocysts (34.6 to 43.2 x 20.2 to 30.0  $\mu$  m) with a length:width ratio of 1.40 to 1.73. *I. hippopotami* n.sp. from *Hippopotamus amphibius* has ovoid to broadly ellipsoidal oocysts, no micropyle, polar cap, oocystic residuum or polar granules, subspherical to ovoid **sporocyst** residuum present as clumps and 4 elongated pear-shaped sporozoites. **Eimeria** *ailuri* n.sp. from *Ailurus fulgens* has ellipsoidal oocysts, no micropyle, polar cap, oocystic residuum or polar granules, has ellipsoidal **sporocyst** with a small stieda body at the narrower end and **sporocystic** residuum present as an aggregation of dark granules in the centre. Apart from *I. pantheri* and *I. mohini*, all the other new species represent new host records for coccidia. 4 ref.

3/7/13 (Item 8 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00639669 CAB Accession Number: 780847102  
**Eimeria** *gundii* n.sp. (Protozoa: Eimeriidae) from Tunisian gundi (*Ctenodactylus gundi*).  
Mishra, G. S.; Gonzalez, J. P.  
Pasteur Inst., Tunis, Tunisia.  
Annales de Parasitologie Humaine et Comparee vol. 53 (2): p.241-243  
Publication Year: 1978  
ISSN: 0003-4150 --  
Language: English  
Document Type: Journal article  
Of 17 *Ctenodactylus gundi* caught in southern Tunisia, 11 had coccidial

oocysts in their faeces and, at autopsy, in the intestinal mucosa. 20 oocysts measured 20 to 27 by 18 to 23  $\mu\text{m}$  (average 23.6 by 20.3  $\mu\text{m}$ ). The oocyst wall was 1.0 to 1.5  $\mu\text{m}$  thick, no micropyle was seen, there was no polar granule and the oocyst residuum measured 8 to 10  $\mu\text{m}$ . The **sporocysts** were 9 to 12 by 5 to 7  $\mu\text{m}$  (average 10.6 by 6.6  $\mu\text{m}$ ); there was a stieda body at the narrower end of the **sporocyst** and the sporozoites, which measured 6 to 8 by 2.5 to 3  $\mu\text{m}$ , surrounded a small compact granular residuum. This is the first report of an **Eimeria** species in the gundi. It is named **Eimeria gundii** n.sp. ADDITIONAL ABSTRACT: In Tunisia, nematode and cestode **eggs** were found in the faeces of all of 17 Ctenodactylus gundi. Trichostrongylidae, Trichuridae, Oxyuridae and Hymenolepididae were found in the intestines at post-mortem examination.

3/7/14 (Item 1 from file: 77)  
 DIALOG(R)File 77:Conference Papers Index  
 (c) 2002 Cambridge Sci Abs. All rts. reserv.

4614636  
 Supplier Accession Number: 01-06979 V29N06  
 Infectivity of E. acervulina oocysts, sporocysts and sporozoites with in ovo delivery  
 Doelling, V.W.; Martin, A.; Hutchins, J.E.; Tyczkowski, J.K.  
 8th International Coccidiosis Conference 0005692 Cairns (Australia)  
 9-13 Jul 2001  
 Molecular Parasitology Unit (University of Technology, Sydney),  
 Australian Society for Parasitology  
 University of Technology, Sydney, Department of Cell and Molecular  
 Biology, Westbourne St, Gore Hill NSW 2065, Australia; phone:  
 61-2-9514-4063; fax: 61-2-9514-4026. Poster Paper  
 Languages: ENGLISH  
 ? ds

Set	Items	Description
S1	1547	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
? s s2 and (immuniz? or vaccin? or inject?)		
Processed 20 of 26 files ...		
Processing		
Completed processing all files		
	715	S2
	429407	IMMUNIZ?
	701738	VACCIN?
	2286324	INJECT?
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
? s s4 not s3		
	18	S4
	14	S3
S5	17	S4 NOT S3
? t s5/7/all		
>>>Format 7 is not valid in file 143		

5/7/1 (Item 1 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

12582770 BIOSIS NO.: 200000336272  
 Localization and immunogenicity of a low molecular weight antigen of  
**Eimeria tenella**.  
 AUTHOR: Tennyson Shan A; Barta John R(a)  
 AUTHOR ADDRESS: (a)Department of Pathobiology, Ontario Veterinary College,  
 University of Guelph, Guelph, ON, N1G 2W1\*\*Canada

JOURNAL: Parasitology Research 86 (6):p453-460 June, 2000  
MEDIUM: print  
ISSN: 0932-0113  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English  
SUMMARY LANGUAGE: English

ABSTRACT: A low molecular weight (LMW) antigen recognized by a murine monoclonal antibody (C34F1) was localized within endogenous stages of **Eimeria tenella** (USDA strain 80). Using indirect fluorescent antibody assay and immunoelectron microscopy, the LMW antigen was found in: sporozoites, first, second and third generation meronts, gamonts, unsporulated oocysts, and **sporocysts**. The antigen was observed in the cytoplasm and pellicle of the parasite, and in the parasitophorous vacuole, **sporocyst** walls and cytoplasm of infected host cells. The immunogenicity of this LMW antigen was assessed by antigen-specific serum antibody responses in chickens orally inoculated with live oocysts or **injected** intramuscularly with dead sporozoites. LMW antigen-specific serum antibodies were detected using Western blots of **E. tenella** sporozoites as early as 4 days after sporozoite **injection** and 6 days after oocyst inoculation. Unusually, the monoclonal antibody C34F1 reduced the binding of immune chicken serum to the antigen in a competitive antibody binding assay, but not the reverse, suggesting that there is a single, immunodominant epitope on this antigen.

5/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08845152 BIOSIS NO.: 199395134503  
Ultrastructural observations of host-cell invasion by sporozoites of **Eimeria papillata** in vivo.  
AUTHOR: Chobotar Bill(a); Danforth Harry D; Entzeroth Rolf  
AUTHOR ADDRESS: (a)Dep. Biol., Andrews Univ., Berrien Springs MI 49104\*\*USA  
JOURNAL: Parasitology Research 79 (1):p15-23 1993  
ISSN: 0932-0113  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Scanning and transmission electron microscopy were used to study the invasion of mouse small-intestinal epithelium by sporozoites of **Eimeria papillata**. Some mice received oocysts by gavage and others received either **sporocysts** or sporozoites by direct **injection** into the small intestine. The highest concentration of invaded cells were found in ligated intestinal tissues studied at 5-45 min after the inoculation of sporozoites. Sporozoites actively invaded anterior end first, which resulted in extensive damage to the host cell. Such cells showed disrupted microvilli; protuberances of cytoplasm into the lumen, apparently the result of a disrupted plasma membrane; vacuolization of the cytoplasm; and damage to the mitochondria. These damaged cells were rapidly vacated as the sporozoite moved laterally into one or more adjacent intact host cells without entering the lumen. It is suggested that the host cell initially entered from the lumen becomes so severely traumatized that the parasite of necessity enters an adjacent cell as a prelude to further development. Various aspects of host-cell invasion by coccidia and malarial parasites are reviewed.

5/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07015693 BIOSIS NO.: 000089107577

SELECTION AND CHARACTERIZATION OF A PRECOCIOUS LINE OF **EIMERIA**

-**INTESTINALIS** AN INTESTINAL RABBIT COCCIDIUM

AUTHOR: LICOIS D; COUDERT P; BOIVIN M; DROUET-VIARD F; PROVOT F

AUTHOR ADDRESS: I.N.R.A., LAB. DE PATHOL. DU LAPIN, CENT. DE RECHERCHES DE  
TOURS-NOUZILY, 37380 MONNAIE, FR.

JOURNAL: PARASITOL RES 76 (3). 1990. 192-198. 1990

FULL JOURNAL NAME: Parasitology Research

CODEN: PARPE

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: A precocious line of **Eimeria** intestinalis was obtained by selection for early development of oocysts in rabbits and after six consecutive passages in animals. this line (EiP) was derived from a wild strain (EiO) isolated in 1975 from the caecal content of rabbit with caccidiosis. The prepatent period of the EiP strain was reduced from 215 h to < 144 h, the result being that the oocyst sporulation time was the same for both lines. The excreted and unsporulated oocysts had exactly the same shape, but microscopical examination of the sporulated oocysts showed a marked difference between EiP and EiO strains. A huge refractile globule was located in each of two **sporocysts** of the precocious line, whereas no refractile globule was seen in the other two. The EiP line had a reproductive potential much lower (1000 times) than that of its parent strain EiO and, as judged by the weight gain, mortality and lesions that also occurred in the jejunum and above all in the ileum, its pathogenicity was substantially reduced.

5/7/4 (Item 4 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

06159456 BIOSIS NO.: 000085122608

ANTIBODY DEVELOPMENT AND CELLULAR IMMUNE RESPONSES IN SHEEP **IMMUNIZED**

AND CHALLENGED WITH SARCOCYSTIS-**TENELLA** SPOROCYSTS

AUTHOR: O'DONOGHUE P J; WILKINSON R G

AUTHOR ADDRESS: CENT. VET. LAB., DEP. AGRIC., FROME ROAD, ADELAIDE 5000,  
SOUTH AUSTRALIA.

JOURNAL: VET PARASITOL 27 (3-4). 1988. 251-266. 1988

FULL JOURNAL NAME: Veterinary Parasitology

CODEN: VPAPD

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: Four specific-pathogen-free (SPF) sheep were experimentally infected with 103 or 104 Sarcocystis **tenella** (syn. *S. ovis*) **sporocysts** and another two sheep served as uninfected controls. All sheep were challenged 49 days later by infection with 2.5 .times. 105 **sporocysts** and their humoral and cellular responses to infection and challenge were assessed weekly by enzyme immunoassays and lymphocyte transformation assays. The control sheep died from acute sarcocystosis 29-30 days after challenge, whereas the **immunized** sheep survived and were protected against acute disease. Specific IgM and IgG antibodies were detected in the **immunized** sheep from 28 days after infection onwards. Lymphocytes collected before and after challenge did not exhibit any significant differences in their responses to stimulation with *S. tenella* cystozoite or sporozoite antigens. Furthermore, lymphocytes collected before challenge did not differ from the controls in their responses to stimulation with the mitogens lipopolysaccharide or phytohaemagglutinin. However, lymphocytes collected after challenge did exhibit increased blastogenic responses to stimulation with both mitogens from 21-28 days after challenge onwards. The infected sheep were necropsied 46 days after challenge, and histological and ultrastructural

studies revealed numerous infiltrates of lymphocytes, histiocytes and plasma cells in the skeletal muscles, sometimes in association with degenerating parasitic cysts and macrophage myophagia. Parasites were not completely eliminated nor prevented from further establishment, therefore the protective immunity was not sterile but rather a state of premunition.

5/7/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

05183726 BIOSIS NO.: 000082024347  
ULTRASTRUCTURAL LOCALIZATION OF IMMUNOGLOBULIN A AND IMMUNOGLOBULIN G  
RECEPTORS ON OOCYSTS **SPOROCYSTS** SPOROZOITES AND MEROZOITES OF  
**EIMERIA**-FALCIFORMIS  
AUTHOR: WHITMIRE W M; SPEER C A  
AUTHOR ADDRESS: VETERINARY RESEARCH LAB., MONTANA STATE UNIV., BOZEMAN, MT,  
USA 59717.  
JOURNAL: CAN J ZOOL 64 (3). 1986. 778-784. 1986  
FULL JOURNAL NAME: Canadian Journal of Zoology  
CODEN: CJZOA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The localization of parasite-specific IgA and IgG immunoglobulins on **Eimeria** falciformis oocysts, **sporocysts**, sporozoites, and merozoites was examined by immunoelectron microscopy. Parasites were fixed in glutaraldehyde, incubated with heat-inactivated sera or gut contents from normal or specifically **immunized** mice, reacted with ferritin-conjugated or colloidal gold-conjugated sheep or goat antimouse IgA or IgA antibody and prepared for transmission electron microscopy. Other purified samples of sporozoites or merozoites were exposed to sera or gut contents, fixed in 0.15% glutaraldehyde, and then incubated with ferritin-conjugated or colloidal gold-conjugated sheep or goat antimouse antibody. Parasite-specific IgA and IgG receptors were detected on the plasmalemma of sporozoites and merozoites. Specific IgG receptors were also present on the inner and outer layers of the oocyst wall, and on the inner surface of the **sporocyst** wall. Live sporozoites and merozoites shed immune complexes at their posterior ends. No internal alternations were detected ultrastructurally in sporozoites or merozoites treated with parasite-specific IgA or IgG antibodies.

5/7/6 (Item 6 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04739701 BIOSIS NO.: 000080042828  
EFFECTS OF INTESTINAL CONTENTS FROM NORMAL AND **IMMUNIZED** MICE ON  
SPOROZOITES OF **EIMERIA**-FALCIFORMIS  
AUTHOR: DOUGLASS T G; SPEER C A  
AUTHOR ADDRESS: DEP. MICROBIOL., PUBLIC HEALTH, MICH. STATE UNIV., EAST  
LANSING, MICH.  
JOURNAL: J PROTOZOL 32 (1). 1985. 156-163. 1985  
FULL JOURNAL NAME: Journal of Protozoology  
CODEN: JPROA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The interaction of *E. falciformis* sporozoites with the intestinal epithelium and with the intestinal contents from the cecum and colon of normal and specifically **immunized** mice was studied by light (LM) and scanning electron (SEM) microscopy. Fecal (FM) and enterocyte-associated (EAM) mucus were removed from the cecum and colon

of normal mice and mice that had been **immunized** 1, 6, 12, or 20 days earlier with a series of oral inoculations of *E. falciformis* oocysts. Sporozoite-specific IgA, but neither IgM nor IgG, was detected by the immunofluorescent antibody test in FM and EAM from **immunized** mice. No sporozoite-specific immunoglobulin was detected in normal mice. When examined by LM, sporozoites exposed to all FM and EAM preparations exhibited greater motility and excystation from **sporocysts**. At 4 h after incubation in FM or EAM from normal or immune mice, approx. 10% of the sporozoites appeared damaged, being nonmotile and nonrefractile. Immune FM and EAM caused agglutination of sporozoites and **sporocysts** and oocysts walls of *E. falciformis*, but did not agglutinate those of *E. ferrisi*. Scanning electron microscopy of in vitro interactions between *E. falciformis* sporozoites and intestinal contents revealed that sporozoites exposed to immune EAM were coated with particulate material whereas those exposed to normal EAM were relatively clean. Sporozoites exposed to immune FM were usually embedded within the mucus whereas those exposed to normal FM were situated on top of the mucus. No significant differences occurred between the length/width (L/W) ratios of sporozoites incubated in normal FM and EAM or in PBS. Sporozoites exposed to immune FM had significantly greater L/W ratios than those exposed to normal FM whereas those exposed to immune EAM had significantly shorter L/W ratios than ones exposed to normal EAM. Few of the sporozoites observed on the luminal surface of the colon and cecum of normal mice were covered by mucus and none were altered in shape or showed pellicular damage. Only a few sporozoites were observed on the luminal surface of the colon and cecum of **immunized** mice. Most of these were covered by mucus and some exhibited pellicular alterations.

5/7/7 (Item 7 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

02196331 BIOSIS NO.: 000064038850  
DEMONSTRATION OF CIRCULATING ANTIBODIES TO **EIMERIA-TENELLA** BY  
THE INDIRECT IMMUNO FLUORESCENT ANTIBODY TEST USING SPOROZOITES AND 2ND  
STAGE SCHIZONTS AS ANTIGEN  
AUTHOR: KOUWENHOVEN B; KUIL H  
JOURNAL: VET PARASITOL 2 (3). 1976 (RECD 1977) 283-292. 1976  
FULL JOURNAL NAME: Veterinary Parasitology  
CODEN: VPARD  
RECORD TYPE: Abstract

ABSTRACT: In the indirect immunofluorescent antibody (IFA) test using sporozoites as an antigen, sera from chickens **immunized** via the natural route were positive in dilutions as high as 1:2048. Serum from a rabbit, **immunized** only to sporozoites by s.c. **injection**, was positive in a dilution of 1:4096. Non-**immunized** chicken and rabbit sera were positive in dilutions varying from 1:20-1:64. Sporozoites within **sporocysts** were not stained. In frozen sections of infected chorioallantoic membranes and ceca, sporozoites were not traced with the IFA test with **immunized** chicken or rabbit sera. With the rabbit serum specific diffuse intraepithelial and subepithelial fluorescence was observed in the ceca from 4-11 h after infection. Fluorescence was never associated with the 1st-stage schizonts and gametes, but 2nd-stage schizonts were positive with chicken and rabbit serum. The titers obtained with this antigen were about the same as those obtained with sporozoite smears. The possible presence of common antigens in sporozoites and 2nd stage schizonts is discussed.

5/7/8 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

02095724 Genuine Article#: KA596 Number of References: 26  
Title: DEVELOPMENT AND CHARACTERIZATION OF MONOCLONAL-ANTIBODIES TO  
1ST-GENERATION MEROZOITES OF **EIMERIA**-BOVIS  
Author(s): HAEBER PJ; LINDSAY DS; BLAGBURN BL  
Corporate Source: AUBURN UNIV, COLL VET MED, DEPT PATHOBIOL/AUBURN//AL/36849;  
AUBURN UNIV, COLL VET MED, DEPT PATHOBIOL/AUBURN//AL/36849  
Journal: VETERINARY PARASITOLOGY, 1992, V44, N3-4 (OCT), P321-327  
ISSN: 0304-4017  
Language: ENGLISH Document Type: NOTE

Abstract: Merozoites of **Eimeria** bovis were harvested from bovine monocyte cell cultures and used to **immunize** BALB/C mice. Spleens from **immunized** mice were removed and the cells fused with mouse myeloma cells. Supernates from resulting hybridoma cell lines were examined for antibodies to first-generation E. bovis merozoites using an indirect immunofluorescent antibody (IFA) assay. Three positive cell lines were identified and cloned by limiting dilution. All three cell lines produced immunoglobulins of the IgG1 isotype that recognized antigens in the anterior half to two-thirds of the merozoites. Specificity of the monoclonal antibodies was examined with the IFA assay against sporozoites of E. bovis, sporozoites and merozoites of **Eimeria** papillata from mice and **Eimeria** tenella from chickens, sporozoites of Isospora suis from pigs, and tachyzoites of Toxoplasma gondii and Neospora caninum from cell cultures. Monoclonal antibodies from the three clones reacted with the anterior end of E. bovis sporozoites, but did not react with the other parasites examined. None of the monoclonal antibodies reacted with merozoite antigens in immunoblots.

5/7/9 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03362946 CAB Accession Number: 970802085  
Comparison of the genomic fingerprints generated by the random amplification of polymorphic DNA between precocious lines and parental strains of **Eimeria** spp. from the rabbit.  
Cere, N.; Licois, D.; Humbert, J. F.  
INRA, Laboratoire de Pathologie du Lapin, Station de Pathologie Aviaire et de Parasitologie, F-37380 Nouzilly, France.  
Parasitology Research vol. 83 (3): p.300-302  
Publication Year: 1997  
ISSN: 0044-3255 --  
Language: English  
Document Type: Journal article

Precocious strains of **Eimeria** spp. which are being investigated as possible live **vaccines** for coccidiosis in rabbits are characterized by large refractile bodies in their **sporocysts**, which suggest genetic differences from parental strains. The random amplified polymorphic DNA (RAPD) technique was used to compare the genome of precocious and wild-type strains of E. intestinalis and E. magna from rabbits. Although 80 primers were used alone and in combination, no differences were detected in the patterns of precocious and parental strains of both species. It is suggested that only a small number of genes is involved in the trait for precociousness. 18 ref.

5/7/10 (Item 2 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01739579 CAB Accession Number: 862279080  
Protection against a lethal dose of Sarcocystis **tenella** **sporocysts** by pre-exposure of sheep to a sub-lethal dose.  
Cole, D. J. W.; Jonas, W. E.



New Zealand Journal of Zoology vol. 12 (3): p.443  
Publication Year: 1985  
ISSN: 0301-4223 --  
Language: English  
Document Type: Abstract only

5/7/11 (Item 3 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01732981 CAB Accession Number: 860834244  
Ultrastructural localization of IgA and IgG receptors on oocysts, **sporocysts**, sporozoites and merozoites of **Eimeria falciformis**.  
Whitmire, W. M.; Speer, C. A.  
Vet. Res. Lab., Montana State Univ., Bozeman, MT 59717, USA.  
Canadian Journal of Zoology vol. 64 (3): p.778-784  
Publication Year: 1986  
ISSN: 0008-4301 --  
Language: English Summary Language: french  
Document Type: Journal article

The localization of parasite-specific IgA and IgG on *E. falciformis* oocysts, **sporocysts**, sporozoites and merozoites was examined by immunoelectron microscopy. Parasites were fixed in glutaraldehyde, incubated with heat-inactivated sera or gut contents from normal or specifically **immunized** mice, reacted with ferritin conjugated or colloidal gold-conjugated sheep or goat anti-mouse IgA or IgG antibody and prepared for transmission electron microscopy. Other purified samples of sporozoites or merozoites were exposed to sera or gut contents, fixed in 0.15% glutaraldehyde, and then incubated with ferritin-conjugated or colloidal gold-conjugated sheep or goat anti-mouse antibody. Parasite-specific IgA and IgG receptors were detected on the plasmalemma of sporozoites and merozoites. Specific IgG receptors were also present on the inner and outer layers of the oocyst wall and on the inner surface of the **sporocyst** wall. Live sporozoites and merozoites shed immune complexes at their posterior ends. No internal alterations were detected ultrastructurally in sporozoites or merozoites treated with parasite-specific IgA or IgG antibodies. 13 ref.

5/7/12 (Item 4 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01034483 CAB Accession Number: 800882822  
Sheep sarcosporidiosis: controlling sarco. Interruption of transmission by farm predators and experimental induction of immunity to *Sarcocystis tenella* in lambs.  
Lightowlers, M. W.; Ford, G. E.  
Vet. Parasit. Group Lab., Inst. of Med. & Vet. Sci., Frome Rd., Adelaide, South Australia.  
Conference Title: Australian Society for Parasitology: Programme and abstracts of papers presented at the 24th Conference of the Society, held at Flinders University, South Australia, 19-21 May 1980.  
p.(10)  
Publication Year: 1980  
Publisher: -- ., Australia  
Language: English  
Document Type: Miscellaneous  
Sarcocystis gigantea cystozoites, irradiated *S. tenella* **sporocysts** or *Brucella abortus* lipopolysaccharide in Freund's Complete Adjuvant were used as **vaccine**.

5/7/13 (Item 5 from file: 50)

DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01029544 CAB Accession Number: 800876153

Some studies on **Eimeria tenella** in chickens.

El-Refaie, M. R.

Journal of the Egyptian Veterinary Medical Association vol. 36 (1):

p.95-98

Publication Year: 1976

ISSN: 0379-3044 --

Language: English

Document Type: Journal article

**Eimeria tenella** oocysts were detected in the droppings of chickens one hour after oral inoculation with a million oocysts. Examination of droppings 1.25h after inoculation revealed oocysts with active sporozoites outside **sporocysts** and many free active sporozoites outside the oocysts. Oocysts were passed for up to 5 h. When one ml of immune serum (from birds infected one month previously) was **injected** daily into susceptible chicks there was a reduction in oocyst output. 6 ref.

5/7/14 (Item 6 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

00579548 CAB Accession Number: 772502563

Immunity to coccidiosis: interactions in vitro between **Eimeria tenella** and chicken phagocytic cells.

Rose, M. E.; Long, P. L.

Houghton Poultry Res. Sta., Houghton, Huntingdon, Cambs., UK.

Biochemistry of parasites and host-parasite relationships.

p.449-455

Publication Year: 1976

Editors: Van den Bossche, H.

Publisher: -- Amsterdam, The: North-Holland Publishing Company.,  
Netherlands

Language: English

Document Type: Miscellaneous

Phagocytic cells (polymorphonuclear leucocytes and macrophages), from chickens **immunized** with **Eimeria tenella** adhere to and phagocytose **sporocysts** and sporozoites of the homologous organism more actively than do cells from susceptible chickens. To determine whether this increased activity of the host cells has any significance in protective immunity, the viability of sporozoites incubated in peritoneal exudate cells from normal and **immunized** chickens was estimated. Sporozoites were inoculated into developing chicken embryos and the infections induced were measured by mortality and haemorrhage, or by oocyst production. No differences were found, suggesting that, although phagocytic cells from specifically **immunized** chickens interact more readily with sporozoites of **E. tenella**, they are not, per se, capable of affecting subsequent development of the parasite. These results, together with those from work in progress, suggest that additional factors, either antibodies and/or the products of the interactions of sensitized lymphocytes with antigens, may be necessary for the full expression of immunity. (AS).

5/7/15 (Item 7 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

00106729 CAB Accession Number: 732208844

Biochemical investigation with regard to infection and immunity of **Eimeria acervulina** in the fowl.

Horst, C. J. G. van der; Kouwenhoven, B.  
Clinic Vet. Obstetrics, Yalelaan 7, Utrecht, Netherlands.  
Zeitschrift fur Parasitenkunde vol. 42 (Heft 1): p.23-38  
Publication Year: 1973 --  
Language: English

Document Type: Journal article

Biochemical investigation of the fluid surrounding the **sporocysts** in the oocysts ('oocyst fluid') showed the presence of the common amino acids, beta-isoaminobutyric acid, glycerol, an unidentified carbohydrate and proteins. Incubation experiments with labelled glucose revealed the presence of enzymes able to convert glucose into lactic acid and other acids. Inside the **sporocysts** the common amino acids, glycerol and the unidentified carbohydrate were also present, but beta-isoaminobutyric acid did not occur. The carbohydrate binding protein 'carboglutelin', carbohydrate phosphate and small amounts of glucose and fructose were mainly found inside the **sporocysts**. Incubation experiments of intestinal pieces both from **immunized** and non-**immunized** birds with oocyst fluid, **sporocysts** and labelled glucose showed that a stronger reaction took place in **immunized** birds than in those not **immunized** ones. Similar experiments were performed with non-**immunized** birds at different days after a primary infection. The reaction of the intestinal wall, which seemed quite normal again 19 days after infection, was comparable with that observed in **immunized** birds. It is postulated that in the first part of the intestine of **immunized** birds some compounds are present originating from the first infection. These compounds might enhance the reaction between the oocyst fluid and glucose to such an extent that the pH decreases and epithelial cells are pushed off possibly together with the **sporocysts**. Then leakage of serum proteins might occur.

5/7/16 (Item 8 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00106728 CAB Accession Number: 732208843

Histological observations with respect to the immune mechanism in **Eimeria acervulina** infection in the domestic fowl.

Kouwenhoven, B.; Horst, C. J. G. van der  
Poult. Hlth Serv., Oude Rijkssstraatweg 43, Doorn, Netherlands.  
Zeitschrift fur Parasitenkunde vol. 42 (Heft 1): p.11-21  
Publication Year: 1973 --  
Language: English

Document Type: Journal article

A strong repulsion of epithelial tissue into the intestinal lumen associated with a lowered intestinal pH, thin liquid intestinal contents and leakage of serum protein into the lumen was observed in **immunized** birds, some hours after oral reinfection. The extruded epithelial tissue degenerated and died in the lumen; massive numbers of dying cells could also be observed in tissue sections of the intestinal contents. Most likely the sporozoites are pushed off together with the cells. The repulsion was always associated with a prominent swelling of the muscles in the intestinal villi. In the non-immune birds, however, some cell repulsion and a little swelling of the villus muscles was observed, but only at the tips of the villi. In normal uninfected birds the individual contraction of the villus muscles supports the concept of a physiological repulsion of epithelial cells at the so called 'extrusion zone'. In the **immunized** birds the reaction of the tissue after reinfection could be completely suppressed by cortisone treatment. Incubation experiments of intestinal pieces both from **immunized** and nonimmunized birds with oocyst fluid, **sporocysts** and glucose showed similar reactions; the reaction was much stronger in **immunized** than non-**immunized** birds. These results indicate a local tissue immunity. It is postulated that after a primary infection some compounds stay behind in the intestinal wall and these can enhance the reaction of oocyst fluid

and **sporocysts** with glucose to such an extent that much lactic acid is formed, which in turn might affect the intestinal wall, resulting in repulsion of the epithelial tissue.

5/7/17 (Item 1 from file: 94)  
DIALOG(R)File 94:JICST-EPlus  
(c)2002 Japan Science and Tech Corp(JST). All rts. reserv.

01378198 JICST ACCESSION NUMBER: 91A0509187 FILE SEGMENT: JICST-E  
Site Specificity of **Eimeria Tenella** and **Eimeria** Maxima in Chickens.

SHIOTANI NAOE (1); NAKANISHI TERUO (1)

(1) Osaka Joshigakuen Junior College

Osaka Joshi Gakuen Tanki Daigaku Kiyo(Bulletin of Osaka Joshigakuen Junior College), 1990, NO.34, PAGE.9-17, TBL.5, REF.17

JOURNAL NUMBER: S0784BAA ISSN NO: 0286-0570

UNIVERSAL DECIMAL CLASSIFICATION: 619:616.9 636.52/.58

LANGUAGE: Japanese COUNTRY OF PUBLICATION: Japan

DOCUMENT TYPE: Journal

ARTICLE TYPE: Original paper

MEDIA TYPE: Printed Publication

ABSTRACT: Distribution of oocysts, **sporocysts** and sporozoites of **Eimeria tenella** and **Eimeria** maxima in the digestive tract of chickens was investigated. **Sporocysts** came out immediately after the oral inoculation of oocysts. At 1 hour after the inoculation, approximately 75% of total **E.tenella sporocysts** were already present in the ceca. Thereafter the number of **E.tenella sporocysts** decreased gradually. **E.tenella** sporozoites in the ceca increased and kept high level until 12 hours after the inoculation. Small number of **sporocysts** and sporozoites of **E.tenella** were found in other intestinal sites. While a great number of **E.maxima** sporozoites were found especially in the jejunum at 2 hours after the inoculation. Apparent accumulation of **E.maxima sporocysts** was not observed in all sites of the digestive tract. Histological samples were taken from the chickens killed 1, 2 and 3 hours after the **injection**, and examined for the number of invaded sporozoites. At 1 hour after the **injection**, the number of **E.tenella** sporozoites in the cecal mucosa was significantly larger than that in the jejunum. At 2 hours after the **injection** the number in the ceca increased, while the number in the jejunum did not change. The facts that **E.tenella** sporozoites were mainly found in the ceca, comparing with numerous **E.maxima** sporozoites recovered only in the jejunum, and that they invaded the ceca more readily than the jejunum indicate the presence of site specificity of ceca for **E.tenella** and jejunum for **E.maxima** even before the invasion and development take place. (author abst.)

? ds

Set	Items	Description
S1	1547	SPORO CYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S5	17	S4 NOT S3
? s		(Eimeria or tenella or necatrix or acervulina or parecox or brunetti or mitis)
	26120	EIMERIA
	10690	TENELLA
	2421	NECATRIX
	3717	ACERVULINA
	1	PARECOX
	1366	BRUNETTI
	7291	MITIS
S6	36420	(EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX

```

                OR BRUNETTI OR MITIS)
? s s6 and (ovo or egg?)
    36420 S6
    9651 OVO
    655710 EGG?
    S7    984 S6 AND (OVO OR EGG?)
? s s7 and (immuniz? or vaccin? or inject?)
    984 S7
    429407 IMMUNIZ?
    701738 VACCIN?
    2286324 INJECT?
    S8    120 S7 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
? rd s8
...examined 50 records (50)
...examined 50 records (100)
...completed examining records
    S9    72 RD S8 (unique items)
? ds

```

Set	Items	Description
S1	1547	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S5	17	S4 NOT S3
S6	36420	(EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)
S7	984	S6 AND (OVO OR EGG?)
S8	120	S7 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S9	72	RD S8 (unique items)

? s (treat? or disrupt? or grind?) and s9

Processing

Processed 10 of 26 files ...

Processing

Processed 20 of 26 files ...

Completed processing all files

11410893 TREAT?

442342 DISRUPT?

89129 GRIND?

72 S9

S10 19 (TREAT? OR DISRUPT? OR GRIND?) AND S9

? ds

Set	Items	Description
S1	1547	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S5	17	S4 NOT S3
S6	36420	(EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)
S7	984	S6 AND (OVO OR EGG?)
S8	120	S7 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S9	72	RD S8 (unique items)
S10	19	(TREAT? OR DISRUPT? OR GRIND?) AND S9

? t s10/7/all

>>>Format 7 is not valid in file 143

10/7/1 (Item 1 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

10389206 BIOSIS NO.: 199699010351

Livacox, an attenuated **vaccine** against coccidiosis of chickens.  
AUTHOR: Bedrník Petr  
AUTHOR ADDRESS: BIOPHARM, Res. Inst. Biopharmacy Veterinary Drugs,  
CZ-25449 Jilove near Prague\*\*Czech Republic  
JOURNAL: Magyar Allatorvosok Lapja 51 (1):p34-36 1996  
ISSN: 0025-004X  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: Hungarian; Non-English  
SUMMARY LANGUAGE: Hungarian; English

ABSTRACT: **Immunization** against coccidiosis of chickens using the live attenuated Livacox **vaccine** seems to be an appropriate alternative as compared to the use of anticoccidial drugs both in laying and broiler flocks. Livacox **vaccine** contains coccidia of attenuated lines. Their pathogenicity was diminished by adaptation to grow in chicken embryos and serial passages in **eggs** or by a selection for precocious lines. The attenuation proved to be stable in at least five back passages carried out in chickens. One ml of the **vaccine** contains 100 **vaccination** doses which actually consists of 500 oocysts each of 3 coccidium species (*E. tenella*, *E. maxima* and *E. acervulina*) in 1% solution of chloramine-B (trihydro-Na-benzene-sulphochloramide). The **vaccine** proved to be stable for at least 9 months when stored between +2 degree C and 8 degree C. Freezing must to be avoided. Livacox is applied in one single dose via the drinking water. One ml of the **vaccine** should be diluted in approx. one liter of drinking water. The diluted **vaccine** should be given to chickens thirsted two hours before **vaccination**. It is advisable to **vaccinate** between the age of 7 and 10 days of chickens. Between two days before and 7 days after **vaccination**, the birds should be fed with feeds without containing any anticoccidial agent. All kinds of sulfonamide therapy should be stopped at least two days before **vaccination**. Its application is safe, even in case of 10 times overdose, it causes slight alterations in the gut of chickens. A compatibility with other **vaccinations** was not detected. There is no withdrawal time (food hygienic prohibition term) of consumption. Livacox was registered in Czecho-Slovakia in 1992. Of the 12 million doses sold in 1994, 7 million doses were used in broiler production. In that the indices were the same of slightly better than in birds **treated** before by anticoccidial drugs.

10/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10099536 BIOSIS NO.: 199598554454  
The effect of in **ovo** oocyst or sporocyst inoculation on response to subsequent coccidial challenge.  
AUTHOR: Watkins K L(a); Brooks M A; Jeffers T K; Phelps P V; Ricks C A  
AUTHOR ADDRESS: (a)Elanco Animal Health, Lilly Corporate Center, Building 13/4 Drop 2047, Indianapolis, IN 46285\*\*USA  
JOURNAL: Poultry Science 74 (10):p1597-1602 1995  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: A trial was conducted to investigate the effects of in **ovo** *Eimeria* maxima inoculation on response to subsequent posthatch challenge with *E. maxima*. The in **ovo** **treatments** were arranged in a 4 x 2 factorial with four in **ovo** inoculation sites (air cell, amnion, yolk sac, and allantois) and two parasite forms (oocyst and sporocyst). Four control **treatments** included an uninoculated (naive) unchallenged group, a naive challenged group, and two posthatch

inoculated challenged groups. Chicks were challenged by crop incubation with 50,000 sporulated *E. maxima* oocysts 10 d posthatch. On Day 8 postchallenge, feed intake was determined and birds were weighed and lesions scored. During the brooding period, oocysts were isolated from the fecal material of chicks receiving in **ovo** administration of sporocysts in the amnion and sporocysts or oocysts in the yolk sac. Posthatch inoculated chicks had gain and feed:gain ratios similar to those of naive unchallenged chicks. Gain, feed:gain ratio, lesion scores, and oocyst shedding of chicks inoculated in **ovo** were similar to those of naive, challenged chicks. Although there was some indication that parasites introduced in **ovo** may complete their life-cycle within the developing chick, this experiment provided no evidence that in **ovo** administration of either *E. maxima* oocysts or sporocysts will protect birds from subsequent coccidial challenge. Contrarily, inoculating chicks on day of hatch with a single oral dose of *E. maxima* oocysts provided significant protection against subsequent coccidial challenge.

10/7/3 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

06942609 Genuine Article#: 106KY Number of References: 5  
Title: Intestinal protozoa important to poultry  
Author(s): Mcdougald LR (REPRINT)  
Corporate Source: UNIV GEORGIA, DEPT POULTRY SCI/ATHENS//GA/30602 (REPRINT)  
Journal: POULTRY SCIENCE, 1998, V77, N8 (AUG), P1156-1158  
ISSN: 0032-5791 Publication date: 19980800  
Publisher: POULTRY SCIENCE ASSOC INC, 1111 NORTH DUNLAP AVE, SAVOY, IL 61874

Language: English Document Type: ARTICLE

Abstract: Parasites of two groups are important in poultry, the coccidia and the mastigophora (flagellates) (Table 1). Most of the Coccidia in poultry are in the genus **Eimeria**, but a few species of *Isospora*, *Cryptosporidium*, and *Sarcosporidia* are represented. The **Eimeria** are best known, with seven important species recognized in chickens and several others in turkeys. Diagnosis of coccidiosis is by recognition of classic signs and lesions, by gross examination, and can be aided by microscopic examination of feces and intestinal contents. Control of coccidiosis is by preventive use of anticoccidials and by **immunization**. *Cryptosporidium* are common in poultry but little is known of their importance, except for the occasional outbreak of respiratory cryptosporidiosis in turkey poults.

Of the flagellates, *Histomonas meleagridis* is the best known. Infections in turkeys may cause near 100% mortality, but outbreaks in chickens are more often marked by morbidity and subsequent recovery. Recent outbreaks in broiler breeder pullets caused excessive losses from mortality (5 to 20%) culling, and overall poor flock performance. *Histomonas* organisms are carried by **eggs** of the cecal worm *Heterakis gallinarum*, enabling them to survive for long periods in soil as a source of infection. In the U.S. there are no products available for **treatment** of blackhead. Preventive use of anthelmintics to destroy the cecal worm carrier show some promise in reducing exposure.

10/7/4 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

04093945 CAB Accession Number: 20013108528  
Concurrent Meetings of the Southern Poultry Science Society, 22nd Annual Meeting and The Southern Conference on Avian Diseases, 42nd Annual Meeting, 15 - 16 January 2001.  
USA, Poultry Science Association

Conference Title: Concurrent Meetings of the Southern Poultry Science Society, 22nd Annual Meeting and The Southern Conference on Avian Diseases, 42nd Annual Meeting, 15-16 January 2001.

Poultry Science vol. 80 (7): p.1007-1048

Publication Year: 2001

ISSN: 0032-5791 --

Language: English

Document Type: Journal issue; Conference proceedings

The following article compiles a total of 190 abstracts of investigations conducted in the field of poultry science, mainly focusing on nutrition, nutrient requirements, and **egg** and meat production responses to feeding. Different aspects of management, housing and breeding are presented. The other investigations deal with immunology and physiology, as well as elaborate on poultry diseases, and the pathology, diagnosis, **treatment** and prevention or control of these. The slaughter of broilers is discussed in relation to carcass composition and quality, sanitation and food safety. The costs and benefits of new and proposed food safety regulations are assessed. Ultraviolet radiation, ELISA, HPLC, PCR, enzyme immunoassay, haemagglutination inhibition test, and computer-based instructional techniques for poultry production are some of the technological applications cited.

10/7/5 (Item 2 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

03331260 CAB Accession Number: 972201569

The effect of lymphokines from T cells of birds immune to Salmonella enteritidis against **Eimeria tenella**-infection in broilers.

Original Title: Efecto de las linfoquinas provenientes de linfocitos T, de aves inmunes vs Salmonella enteritidis (SE-ILK), ante la infeccion por **Eimeria tenella** en pollos de engorda.

Garcia Espinosa, G.; Tellez Isaias, G.; Casaubon Hugenin, M. T.; Kogut Henri, M.

Departamento de Produccion Animal: Aves, Facultad de Medicina Veterinaria y Zootecnia, Universidad Nacional Autonoma de Mexico, 04510, Mexico D.F., Mexico.

Veterinaria Mexico vol. 27 (3): p.195-199

Publication Year: 1996

ISSN: 0301-5092 --

Language: Spanish Summary Language: english

Document Type: Journal article

15 chicks were **injected** intraperitoneally at the age of 14 days with 1 ml of soluble products from concanavalin A-stimulated T cells from S. enteritidis-infected chickens. Two day later, the birds were given orally 1000 oocysts of E. **tenella**. A second group was challenged similarly with E. **tenella** but without prophylactic **treatment**, and a third group was left as an untreated and unchallenged control. Each trial was replicated twice. All birds were killed 7 days after the challenge. The birds with prophylactic **treatment** had significantly lower caecal gross lesion scores and total faecal **egg** counts. 25 ref.

10/7/6 (Item 3 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

03197424 CAB Accession Number: 962204314

**Eimeria** sp and Strongyloides papillosus infections in sheep during growth and pregnancy.

Original Title: Przebieg inwazji **Eimeria** spp. in Strongyloides papillosus u owiec w okresie rozwoju i ciazy.

Romaniuk, K.; Gaca-Lagodzinska, K.; Sokol, R.; Michalski, M.; Bah, M.



Katedra Parazytologii i Chorob Inwazyjnych, Wydział Weterynaryjny,  
Akademia Rolniczo-Techniczna, 10-957 Olsztyn-Kortowo, Poland.

Medycyna Weterynaryjna vol. 51 (10): p.590-592

Publication Year: 1995

ISSN: 0025-8628 --

Language: Polish Summary Language: english

Document Type: Journal article

In studies of 40 ewes aged 7 months naturally infected with **Eimeria** and **Strongyloides papillosus**, the animals were divided into 4 **treatment** groups of 10 sheep each. Group I was given selenium/vitamin E preparation, group II was **injected** with Systamex (2.265% oxfendazole) at 7.5 mg/kg whereas group III was given both **treatments**. Group IV served as control. All **treatment** regimes were applied at the onset of puberty, before mating, at the first week of gestation, 7 days before parturition and 7 days after weaning. Faecal **egg** counts for all groups following each application are tabulated. Ewes in group III had significantly lower faecal **egg** counts at mating, drying off and during pregnancy. 25 ref.

10/7/7 (Item 4 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

02649778 CAB Accession Number: 932231880

Immune profile of lambs with sub-clinical eimeriosis

Original Title: Parametri ai profilului imunologic la miei cu eimerioza subclínica

Cozma, V.; Spinu, M.; Ognean, L.; Kadar, L.; Negrea, O.; Chirila, F.

Facultatea de Medicina Veterinara, Cluj-Napoca, 3400 Romania.

Buletinul Institutului Agronomic Cluj-Napoca. Seria Zootehnie si Medicina Veterinara vol. 46 p.177-184

Publication Year: 1992 --

Language: Romanian Summary Language: english

Document Type: Journal article

Groups of five lambs with chronic **Eimeria** infection were **treated** with a copper/selenium preparation, (Cuprosel) with a **vaccine** against *Escherichia coli*, or with the 2 **treatments** combined. The Cu/Se **treatment** on its own induced a 100% increase in the excretion of **Eimeria eggs**, while the *E. coli* **vaccine** reduced that excretion. The combination of both **treatments** reduced **Eimeria** infection by 80%. 18 ref.

10/7/8 (Item 5 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

02550280 CAB Accession Number: 920510222

Moxidectin: systemic activity against common cattle grubs (*Hypoderma lineatum*) (Diptera: Oestridae) and trichostrongyle nematodes in cattle.

Scholl, P. J.; Guillot, F. S.; Wang, G. T.

Department of Entomology, Louisiana State University, Baton Rouge, LA 70803, USA.

Veterinary Parasitology vol. 41 (3-4): p.203-209

Publication Year: 1992

ISSN: 0304-4017 --

Language: English

Document Type: Journal article

Moxidectin, a macrocyclic lactone endectocide (derived from the actinomycete *Streptomyces cyaneogriseus noncyanogenus*), was evaluated for its efficacy against the migrating 1st-instar larvae of *H. lineatum* and against nematode (trichostrongyles, *Nematodirus* and *Trichuris* spp.) **egg** production in beef cattle (Hereford heifer calves). It was observed that all 3 levels (0.1, 0.2 and 0.4 mg moxidectin kg<sup>-1</sup>) were 100%

effective against oestrid larvae when administered as a s.c. injection. The same levels of treatment were very effective (90-100%) in reducing trichostrongyle nematode egg production. However, there was a slight indication that at least one species, *Cooperia oncophora*, was not completely eliminated, as it was observed that small numbers of eggs began to appear after 2 weeks post-treatment when there had been no opportunity for reinfection. Moxidectin at all doses tested was ineffective against tapeworms (*Moniezia* spp.) and coccidia (*Eimeria* spp.). 4 ref.

10/7/9 (Item 6 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02025384 CAB Accession Number: 880850713  
Development of salinomycin as an anticoccidial in replacement chickens.  
Dost, G.; Raether, W.  
Hoechst AG, 6230 Frankfurt/Main 80, German Federal Republic.  
Conference Title: Research in avian coccidiosis. Proceedings of the Georgia Coccidiosis Conference, 18-20 November 1985  
p.285-293  
Publication Year: 1986  
Editors: McDougald, L. R.; Joyner, L. P.; Long, P. L  
Publisher: University of Georgia College of Agriculture -- Athens, Georgia 30602, USA  
Language: English  
Document Type: Conference paper  
The value of salinomycin as a coccidiostat was evaluated (in the German Federal Republic) in chicks raised for 8 weeks each in battery and floor pen conditions, followed by a 4-week drug-free period and a 3 months laying period. The tolerance limit of Warren-Isabrown chicks and Lohman selected Leghorn chicks were 120 and 80 ppm salinomycin respectively. This was attributable to the lower feed intake of Warren-Isabrown chicks. At dosages of 30-120 ppm, the 2 breeds ingested respectively 1.09 to 4.50 and 1.25 to 5.87 mg/kg bodyweight of salinomycin/day. Replacement chicks on 30 to 60 ppm salinomycin developed lasting protection against challenge after both natural and experimental infection with 500 to 200 000 *Eimeria* oocysts. The performance (number of eggs, egg mass, feed conversion) of the immunized pullets was considerably better than that of controls during the laying period in battery and floor pen conditions. The fertility and hatchability figures suggested that the results were better when cockerels treated with salinomycin at 60 ppm were used. 6 ref.

10/7/10 (Item 7 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01967864 CAB Accession Number: 880846372  
Studies on the immunogenicity of seven attenuated lines of *Eimeria* given as a mixture to chickens.  
Shirley, M. W.; Millard, B. J.  
Houghton Poultry Res. Sta., Houghton, Huntingdon, Cambs. PE17 2DA, UK.  
Avian Pathology vol. 15 (4): p.629-638  
Publication Year: 1986  
ISSN: 0307-9457 --  
Language: English Summary Language: french; german; spanish  
Document Type: Journal article  
12-day-old chicks were inoculated with precocious lines of the 7 species of *Eimeria* (*E. acervulina*, *E. brunetti*, *E. mitis*, *E. necatrix*, *E. praecox*, *E. maxima* and an egg-adapted line of *E. tenella*) occurring in chickens. A mixture of *Eimeria* spp. in 4% aqueous solution of carboxymethyl cellulose (supplemented with

formaldehyde in some experimental variants) was sprayed onto the surface of food in the pens. The theoretical dosages/bird are given. 28 days after inoculation there was no significant difference in mean body weight of **treated** and untreated chickens. 6 days after challenge the inoculated chickens gained almost as much weight as the unimmunized unchallenged controls (although the small differences in weight gain were significant), whereas the unimmunized challenged birds had lost weight. Storage of attenuated lines for 3 or 6 months at 4 deg C did not reduce their **immunizing** ability. In a further experiment, the mean weight gains of chickens given attenuated lines, or the coccidiostats halofuginone or salinomycin, were not significantly different up to the time of challenge. Very few or no gross lesions were present in inoculated chickens after challenge, except in those chickens given stored oocysts of *E. tenella*, indicating that the **egg**-adapted line of *E. tenella* is not as effective as the precocious lines of the other species. 16 ref.

10/7/11 (Item 8 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01323938 CAB Accession Number: 830806357

Annual Report on research and technical work of the Department of Agriculture for Northern Ireland, 1981.

Northern Ireland, Department of Agriculture  
ix + 313pp.

Publication Year: 1982

Animal helminthology pp. 209, 221, 224, 226, 227

Publisher: -- Belfast,

Language: English

Document Type: Annual report

Examination for *Trichinella spiralis* in pork samples for export to Germany and France has reduced the numbers needed to be tested for the home market. None of 5634 samples for export and 2879 for the home market was positive for *T. spiralis*. Of 12 000 human sera examined by ELISA, 123 had readings outside the range of the normal population and are to be examined further. An unusually early case of acute fluke in sheep is reported: 2000 immature *Fasciola hepatica* were removed from the liver of a yearling sheep in August. Very large (>10 000) burdens of *Capillaria* spp. were found amongst a nye of pheasants reared on an estate. Faecal **egg** counts showed that the condition was widespread. Although anthelmintic **treatment** was effective the owner was urged to change the rearing facilities. At Belfast Zoo, *Dirofilaria immitis* was recovered from a coatimundi that had died suddenly. Examination of faecal samples from 2 gemsbucks, a bluckback and a camel revealed moderate burdens of *Camelostrongylus mentulatus*; a maximum of 37 000 was recorded from one of the gemsbucks. ADDITIONAL ABSTRACT: Brief reports are given of research in various areas, including: influence of lipolytic and proteolytic activity on suitability of milk for processing; thermization of milk; pathogenic staphylococci in milk products; adherence and removal of microorganisms from dairy equipment; pre-cleaning **treatment** of mobile tanks; spreadability of butter; effect of type of forage harvester used when ensiling grass on milk production from the spring-calving cow; effect of pattern of concentrate allocation during winter on milk production from autumn-calving cows; supplementation of grass silage for milk production; mastitis immunity; production of flavoured butter; measurement of fat globule size in milk powders; and heat **treatment** of blood/whey mixtures. (See DSA 43, 4858 for 1979 report.) ADDITIONAL ABSTRACT: *Nosema* only was recorded in 45 of 174 honeybees and as part of a mixed infection in 10. *Babesia divergens* is widespread in the counties of Armagh, Fermanagh and Tyrone. The greatest losses occurred in small and medium dairy herds and small beef herds. Of 4000 clinical cases reported annually, 500 died. Experiments suggested that attenuated *B. divergens* (by passage or irradiation) was unlikely to produce a **vaccine** that was

safe for aged non-immune cattle. Examination of faecal samples from lambs revealed high numbers of coccidial oocysts in 1981; some also had high worm **egg** counts. **Injections** of long acting sulphonamides and anthelmintics were effective but very slow because of the lack of fresh grazing land for the lambs. ADDITIONAL ABSTRACT: From trials testing for resistance to *Globodera rostochiensis* (pathotype Ro1) and *G. pallida* (pathotypes Pa1 and Pa3), increasing numbers of multiple pathotype-resistant clones are emerging. Supplies of true botanic potato seeds obtained from the Commonwealth Potato Collection in Edinburgh, Scotland, will enable 100 accessions to be tested annually at the Nematology Laboratory in Northern Ireland for a range of potato cyst nematode pathotypes. Work is continuing on clarifying the limit of selection, the stability of such selected populations and the genetics of interaction between these populations and *Solanum vernei* hybrids. Of 9 samples of plant and soil material submitted for analysis during the year, *Ditylenchus dipsaci* was found in all the narcissus bulbs and *Aphelenchoides ritzemabosi* in one chrysanthemum. ADDITIONAL ABSTRACT: The Plant Pathology Research Division (175-189) includes reports which are abstracted elsewhere.

10/7/12 (Item 9 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00032376 CAB Accession Number: 720303683

Report 1971.

Canada, Department of Agriculture, Research Branch

Report.

377pp.

Publication Year: 1972

Publisher: -- Ottawa.,

Language: English

Document Type: Annual report

This report covers the activities of 37 research stations, experimental farms, institutes and services in Canada, and includes: Research Station, Kentville, Nova Scotia, pp. 17-27: Breeding, nutrition and culture of apples; apple pest control (*Aphis pomi*, *Panonychus ulmi*); and storage. Research Station, Fredericton, New Brunswick, pp. 29-40: A mechanical apple harvester. Research Station, Saint-Jean, Quebec, pp. 65-71: Control of apple pests (*Rhagoletis pomonella*, *Lygus lineolaris*), pathogens (*Venturia inaequalis*) and nutritional disorders. Research Station, Harrow, Ontario, pp. 78-89: Breeding of apricots, peaches and pears; microbiological studies on peaches; and studies on peach rootstocks. Research Station, Ottawa, Ontario, pp. 91-105: Resistance to apple scab (*Venturia inaequalis*); hardy apple rootstocks, and apple colour improvement, thinning and new cvs. Research Station, Vineland Station, Ontario, pp. 107-15: The persistence of dicofol on peach trees; and the ecology and integrated control of insect pests, nematodes and pathogens with particular reference to peach and apple. Food Research Institute, Ottawa, Ontario, pp. 153-61: Apple inheritance studies and effects of rootstocks on fruit composition. Research Station, Summerland, British Columbia, pp. 339-49: Apple growth studies; apple rootstocks and spacing; irrigation and mineral studies on apple trees; shake-and-catch harvesting of tree fruits; CA storage of peaches; insect pests and pathogens of fruit trees; Red Delicious sports; chemical pruning of apple trees; breakdown in Spartan apples during storage; and self-fertile and compact-growing cherries. ADDITIONAL ABSTRACT: This report covers the activities of 37 research stations, experimental farms, institutes and services and includes: Chemistry and Biology Research Institute, Ottawa, Ontario, pp. 129-40: Storage proteins in rhododendron flower buds. Plant Research Institute, Ottawa, Ontario, pp. 163-173: Evaluation of several ornamentals including snapdragons, zinnias, begonias, statice and *Prunus tenella*; uptake of minerals in turfgrasses in relation to road salt damage; and physiology and germination studies in roses. Research Station, Morden,

Manitoba, pp. 227-232: Breeding and selection of lilac, chrysanthemums and lupins; evaluation of street trees; and seed propagation of lilac and *Prinsepia sinensis*. ADDITIONAL ABSTRACT: The report covers the activities of 37 research stations, experimental farms, institutes and services in Canada and includes: Research Station, Sidney, British Columbia, pp. 335-8: Propagation of grapevines. Research Station, Charlottetown, Prince Edward Island, pp. 7-15: Control of strawberry green-petal disease. Research Station, Kentville, Nova Scotia, pp. 17-27: Breeding, nutrition and culture of lowbush blueberries, cranberries, highbush blueberries and strawberries. Research Station, Fredericton, New Brunswick, pp. 29-40: Control of blueberry pests and the nature of growth substances in blueberry buds. Research Station, Saint-Jean, Quebec, pp. 65-71: Control of insect pests (*Lygus lineolaris*, *Tetranychus urticae*) and pathogens (*Sphaerotheca humuli*) of strawberries; and a planting density trial of raspberries. Research Station, Ottawa, Ontario, pp. 91-105: Breeding of raspberries; and selection trials and control of virus diseases of strawberries. Research Station, Agassiz, British Columbia, pp. 323-30: Mechanical harvesting of raspberries; immunity and fertility studies with raspberries; and fungicide trials in raspberries and strawberries. Research Station, Vancouver, British Columbia, pp. 351-9: Control of virus diseases of highbush blueberry, thornless boysenberry and elderberry.

10/7/13 (Item 1 from file: 98)  
DIALOG(R)File 98:General Sci Abs/Full-Text  
(c) 2002 The HW Wilson Co. All rts. reserv.

04014401 H.W. WILSON RECORD NUMBER: BGS199014401 (THIS IS THE FULLTEXT)  
Heat-shock proteins, molecular chaperones, and the stress response:  
evolutionary and ecological physiology.

AUGMENTED TITLE: review

Feder, Martin E

Hofmann, Gretchen E

Annual Review of Physiology (Annu Rev Physiol) v. 61 ('99) p. 243-82

LANGUAGE: English

COUNTRY OF PUBLICATION: United States

WORD COUNT: 20648

ABSTRACT: Molecular chaperones, including the heat-shock proteins (Hsps), are a ubiquitous feature of cells in which these proteins cope with stress-induced denaturation of other proteins. Hsps have received the most attention in model organisms undergoing experimental stress in the laboratory, and the function of Hsps at the molecular and cellular level is becoming well understood in this context. A complementary focus is now emerging on the Hsps of both model and nonmodel organisms undergoing stress in nature, on the roles of Hsps in the stress physiology of whole multicellular eukaryotes and the tissues and organs they comprise, and on the ecological and evolutionary correlates of variation in Hsps and the genes that encode them. This focus discloses that (a) expression of Hsps can occur in nature, (b) all species have hsp genes but they vary in the patterns of their expression, (c) Hsp expression can be correlated with resistance to stress, and (d) species' thresholds for Hsp expression are correlated with levels of stress that they naturally undergo. These conclusions are now well established and may require little additional confirmation; many significant questions remain unanswered concerning both the mechanisms of Hsp-mediated stress tolerance at the organismal level and the evolutionary mechanisms that have diversified the hsp genes. With permission, from the Annual Review of Physiology, Volume 61, 1999, by Annual Reviews Inc. (<http://www.annurev.org>).

TEXT:

KEY WORDS: hsp, temperature, protein denaturation and folding, inducible tolerance, environmental gradients

INTRODUCTION

Although heat-shock proteins (Hsps) first achieved notoriety as gene products whose expression is induced by heat and other stresses (1, 2), discoveries of the past decade have shifted the focus of research to understanding the roles of Hsps as molecular chaperones (3-5). As a result, Hsps, their close relatives, their molecular partners, and many newly discovered proteins are now known to play diverse roles, even in unstressed cells, in successful folding, assembly, intracellular localization, secretion, regulation, and degradation of other proteins (6); failure of these activities is thought to underlie numerous and important human diseases (7). Nonetheless, many of the questions of the past either remain unanswered, awaiting the development of appropriate experimental tools, or can now be revisited with new insights gleaned from the emerging understanding of molecular chaperones. This review seeks to facilitate the examination or re-examination of Hsps as responses to natural stress in diverse organisms inhabiting environments outside the laboratory, the function of Hsps in tolerance of natural stresses, and ecological and evolutionary variation in the heat-shock system. The review sequentially considers (a) the principal implications of laboratory-based studies for ecological and evolutionary research on Hsps, (b) expression of Hsps in nature, (c) covariation of Hsp expression with environmental and biological gradients of stress intensity, (d) the consequences of Hsp expression for fitness, and (e) evolutionary variation in Hsps and the genes that encode them. The primary objective of this review is to redirect the focus of evolutionary and ecological research on Hsps beyond the conclusions that are now well-established and onto the many important questions that remain unanswered.

#### STATE OF THE LITERATURE ESTABLISHED CONCLUSIONS

The relevant literature on Hsps and molecular chaperones is huge, now comprising more than 12,000 references. Even a review of the relevant reviews is difficult. For this reason, we begin by describing several well established conclusions and cite a few of the many excellent recent reviews at diverse levels of sophistication (4-6, 8-11).

The genes encoding Hsps (hsps) are highly conserved and occur in every species in which they have been sought. Many of these genes and their products can be assigned to families on the basis of sequence homology and typical molecular weight (6): hsp110, hsp100, hsp90, hsp70, hsp60, hsp40, hsp10, and small hsp families. Gething (6) recognizes 7 additional families and 12 genes/proteins for which families have not yet been described. In eukaryotes, many families comprise multiple members that differ in inducibility, intracellular localization, and function.

Hsps function as molecular chaperones; i.e. they interact with other proteins and, in so doing, minimize the probability that these other proteins will interact inappropriately with one another. Hsps recognize and bind to other proteins when these other proteins are in non-native conformations, whether due to protein-denaturing stress or because the peptides they comprise have not yet been fully synthesized, folded, assembled, or localized to an appropriate cellular compartment. Binding and/or release of these other proteins is often regulated by association with and/or hydrolysis of nucleotides. Typically, Hsps function as oligomers, if not as complexes of several different chaperones, co-chaperones, and/or nucleotide exchange factors. Interaction with chaperones is variously responsible for (a) maintaining Hsps' partner proteins in a folding-competent, folded, or unfolded state; (b) organellar localization, import, and/or export; (c) minimizing the aggregation of non-native proteins; and (d) targeting non-native or aggregated proteins for degradation and removal from the cell. Presumably, the last two functions are most important in coping with environmental stress.

Not all Hsps are stress-inducible, but those that are respond to a variety of stresses, including extremes of temperature, cellular energy depletion, and extreme concentrations of ions, other osmolytes, gases, and various toxic substances. Activation of various intracellular signaling pathways results in Hsp expression. All known stresses, if sufficiently

intense, induce Hsp expression. Accordingly, Hsps are equally well termed stress proteins, and their expression is termed the stress response. A common aspect of these inducing stresses is that they result in proteins having non-native conformations (12), which is consistent with the function of Hsps as molecular chaperones.

#### IMPLICATIONS OF THE PUBLISHED LITERATURE FOR ECOLOGICAL AND EVOLUTIONARY STUDIES OF HSPS

Space limitations necessitate that we choose among numerous equally valuable references in preparing this review. To present both the breadth and depth of research relevant to the evolutionary and ecological physiology of the heat-shock response, we have compiled a near-comprehensive bibliography of that literature, which is available electronically (13) on the World Wide Web in the Supplemental Materials Section of the main Annual Reviews site (<http://www.AnnualReviews.org>).

A first implication of this massive literature is that the Hsp field has long ago concluded its exploratory phase. Showing that an as-yet-unexamined species expresses Hsps in response to heat or other stresses no longer has any particular novelty.

Second, much of the work on Hsps outside the laboratory or in nonmodel organisms was undertaken before the molecular diversity of Hsps and their function as molecular chaperones was obvious. In the interim, the experimental tools for examining Hsps and the standards for such examinations have both advanced considerably. As a result, much of the earlier work on evolutionary and ecological physiology of Hsps regrettably either does not withstand current scrutiny or contributes little to issues of current interest. Several issues are obvious:

1. Many of the apparently singular Hsps of previous years, often detected by one-dimensional electrophoresis and autoradiography, are now known to represent entire families of Hsps, often with (a) discrete distributions within the cell (e.g. cytoplasmic-nuclear, mitochondrial, chloroplast, or endoplasmic reticulum), (b) different degrees of inducibility (constitutively expressed, constitutively expressed but increasing during or after stress, exclusively inducible), (c) differing kinetics of induction and removal from the cell, and (d) differing tissue specificity. Representing this diversity as a single Hsp or two Hsps ("constitutive" and "inducible") through use of nonspecific probes or lysates of whole organisms and organs can obscure phenomena of great significance (e.g. compare Refs. 14 and 15 with 16). This problem is sometimes remediable only with great difficulty. Often, highly specific probes are available only for standard model organisms, particularly at the level of proteins, and great care must be taken in applying these probes to non-standard organisms (17).

2. Inducible stress tolerance is increasingly understood to result from numerous molecular mechanisms, of which Hsps are collectively only one. Other mechanisms include synthesis of osmotic stress protectants such as polyols and trehalose, modifications of the saturation of cell membrane lipids (homeoviscous adaptation), compensatory expression of isozymes or allozymes of significant enzymes, metabolic arrest, radical scavengers (superoxide dismutase, glutathione system, cytochrome P450), and so on. Accordingly, the unambiguous attribution of stress tolerance to Hsps in general or to any specific Hsp requires more than correlative evidence (18-21). Increasingly, proof resulting from genetic or direct experimental manipulation is becoming the standard for establishing the functional or evolutionary significance of Hsps. Again, this rising standard is often met only with great difficulty in ecological and evolutionary physiological studies, for many of the techniques for genetic and experimental manipulation are not readily applicable to the more ecologically and evolutionarily interesting species.

#### HSP-INDUCING STRESS IN NATURE AND NATURAL INDUCTION OF HSPS

Depending on their geographic locale, organisms in nature risk exposure to temperatures ranging from -100[degree] to more than 100[degree]C, and comparable extremes of chemical and gas concentration, food and water

availability, hydrostatic pressure, radiation, and toxic substances of human origin. Seemingly, Hsp expression should be a common occurrence in nature. In reality, however, movement and/or other behaviors may often enable organisms to avoid Hsp-inducing stress in nature by exploiting equable microhabitats in otherwise stressful environments (22-24). Also, biochemical specializations other than Hsps may stabilize many organisms (or particular stages of their life cycles) so that environmental extremes are not particularly stressful.

Even equable environments can contain Hsp-inducing microhabitats, and even mild stresses can induce Hsps when multiple stresses act in combination. For these reasons, we can assume neither the presence nor the absence of Hsp expression in nature; for that matter, we still do not know in any comprehensive sense whether wild organisms routinely, occasionally, or seldom express inducible Hsps. A growing body of evidence, however, establishes that at least in some circumstances and taxa, Hsp induction is not solely a laboratory phenomenon.

One caution in evaluating the subsequent account is that organisms in nature seldom undergo only one stress at a time. For example, an insect larva undergoing natural heat stress in a rotting fruit may simultaneously experience intense ultraviolet radiation, desiccation, and diverse alcohols and aldehydes, among other stresses. This situation differs from that in most laboratory experimentation, which involves one or a few stresses and makes attribution of Hsp expression to a particular stress in nature more complicated.

#### AQUATIC TEMPERATURE STRESS

Due to the physical characteristics of water, the aquatic environment can be extremely stressful to its inhabitants. In general, the high specific heat and thermal conductivity of water ensure that the majority of aquatic organisms will have body temperatures equivalent to that of their surroundings. Furthermore, the relative thermal homogeneity of aquatic environments can frustrate behavioral avoidance of thermal extremes. Some aquatic ectotherms nonetheless inhabit thermally equable habitats or waters with enough thermal diversity to enable behavioral thermoregulation; our focus is on those species that do not.

In the aquatic environment, habitual exposure to Hsp-inducing thermal stress may be most common in sessile organisms that occur in shallow, stagnant water (e.g. ponds, tidepools, swamps, tidal flats) or in the intertidal zone. Corals, for example, routinely undergo thermal stress that results in bleaching, during which the corals' endosymbionts die. Even modest increases in water temperature of 1-2[degree]C can bleach corals; these temperatures also induce Hsp expression in several species (25, 26). Marine intertidal invertebrates undergo even larger increases in body temperatures during tidal emersion (27-30). For example, during aerial exposure intertidal mussels' body temperatures exceed seawater temperatures by more than 20[degree]C (31), resulting in Hsp expression (29). A similar phenomenon occurs in encysted brine shrimp (*Artemia*) embryos (32, 33). Even relatively mobile aquatic ectotherms such as fish may undergo heat shock in nature (34, 35). For example, gobiid fishes of the genus *Gillichthys* can become trapped in shallow water, which is heated by the sun. Summer-acclimatized fish have higher levels of Hsp90 in brain tissue than do winter-acclimatized fish (34). In addition, the threshold Hsp induction temperature for one species, *G. mirabilis*, is significantly higher in summer than in winter. These data suggest that seasonal variation in water temperature can alter the heat-shock response. More exotic venues for aquatic thermal stress include thermal effluents of power plants, hydrothermal vents, and thermal hot springs, in which temperatures can exceed 100[degree]C (see Hsps of Archaea).

#### TERRESTRIAL TEMPERATURE STRESS

Unlike aquatic environments, terrestrial environments often offer diverse heat sources and sinks and retreats that organisms can exploit to avoid thermal stress. Thus, natural thermal stress and accompanying Hsp



expression in terrestrial environments typically involve limitations in mitigating thermal extremes by movement and conflicts between thermoregulation and other needs. Salamanders, for example, which ordinarily maintain cool temperatures in nature, can inadvertently retreat beneath small sunlit rocks that become warm enough to induce hsp70 mRNA expression (36); by abandoning these rocks to find cooler retreats they may risk immediate desiccation or even warmer temperatures.

The least equivocal case for routine exposure to Hsp-inducing temperature stress is for plants, which cannot change location except as seeds or pollen and can be limited in their ability to adjust heat exchange with the environment (37). Thus, plants in nature can become extremely hot (38). By inference, the entire range of plant heat-shock responses (8, 39) should manifest themselves in nature. Indeed, a small number of case studies document natural Hsp expression (40-45), which can be greatest at times of day or in regions of an individual plant at which temperatures are highest (46). Plant species can differ dramatically, however, in both the magnitude and diversity of the particular Hsps that are expressed during days with especially warm weather (41). Plants should also be prone to natural cold stress (47), which ought to induce expression of Hsps (48-51).

Not surprisingly, therefore, many of the cases of natural thermal stress in animals on land involve animals that live inside or on plants (e.g. 52). *Drosophila* larvae and pupae encounter temperatures exceeding 40[degree]C if the necrotic fruit they infest is in the sun, and express Hsp70 in response (53, 54). Presumably, other animals that cannot escape or offset intense solar heat loads will also express Hsps in nature; this hypothesis awaits systematic study. A unique case concerns desert ants, *Cataglyphis*, which voluntarily undergo body temperatures of >50[degree]C, presumably to escape predators (55). The concentration of Hsp70 family members increases in this species before it naturally encounters high temperatures, as if in anticipation (56).

Terrestrial vertebrates are often especially effective in escaping heat stress, but both they and invertebrates are occasionally hyperthermic during intense physical activity, fever, or to conserved water. In birds and mammals, such hyperthermia activates HSF (the heat-shock transcription factor) and increases the level of Hsps (Hsp cognates, constitutively-expressed Hsps) and Hsps (57-59). Natural hypothermia of animals can be far more conspicuous than natural heat stress, involving diapause, overwintering in exposed sites, hibernation, and sometimes outright freezing. Diverse insects express Hsps in response to cold shock or during overwintering in diapause, although the identity of these Hsps, their tissue specificity, and their developmental regulation vary greatly (60-64). Some euthermic rodents express 70-kDa Hsps in response to cold ambient temperatures, possibly in tandem with nonshivering thermogenesis (65), and ground squirrels (*Spermophilus*) increase Hsp70 family members and ubiquitin-protein conjugates during hibernation (66).

In summary, laboratory studies of the heat-shock response often have proceeded far in advance of fieldwork that establishes an ecological context for their interpretation. Documentation of both natural thermal stress and Hsp expression in nature can provide this context, and a small but growing number of field studies demonstrate that such documentation is feasible.

#### INDUCING STRESSES OTHER THAN TEMPERATURE

Virtually every nonthermal stress can induce Hsps (10, 67). Rarely, however, are these nonthermal stresses ecologically relevant; the literature in this area typically focuses on chemical stressors, and the corresponding data are essentially pharmacological. Even when the stress in question is ecologically relevant, few studies of multicellular eukaryotes examine it in the field or in intact tissues and organisms. Some exceptional work, however, concerns plants and brine-shrimp (*Artemia*). The resurrection plant, a desert species, expresses Hsps in vegetative tissues during water stress; this expression is thought to contribute to desiccation tolerance (68). Similarly, rice seedlings express two proteins in the Hsp90 family upon exposure to water stress and elevated salinity

(69). Embryos of the brine shrimp, one of the most hypoxia-tolerant metazoans, contain large quantities of p26, a molecular chaperone hypothesized to stabilize proteins during long bouts of anaerobic dormancy (see Development). Clearly, additional evolutionary physiological research in this area is sorely needed.

A recurrent theme is that thermal stress and these alternative stressors often result in different patterns of Hsp expression, indicating a diversity of regulatory mechanisms. Examples include variation in the expression of Hsp70 and ubiquitin in the *Drosophila* central nervous system under anoxia (70), and in protein expression during osmotic shock in isolated fish gill cells (71).

#### BIOINDICATORS

Owing to its responsiveness to diverse forms of stress, the heat-shock response has undergone widespread application in biomonitoring and environmental toxicology (72-75). In many cases, Hsps are especially useful biomarkers because their induction is much more sensitive to stress than traditional indices such as growth inhibition. The use of Hsps as biomarkers is most widespread in aquatic toxicology. Most of the literature demonstrates elevated Hsp levels or induction of Hsps under laboratory conditions and then proposes Hsps as a potential indicator of pollutants or toxins in the environment. For example, exposing freshwater sponges to pollutants extracted from river water elevates Hsp70 levels, which increase still further when thermal stress is also imposed (76). Additional examples of Hsp expression in aquatic toxicology concern rotifers, (77), marine sponges (78), amphipods (79), polychaetes (80), mollusks (81-84), and fish (85-87). Other applications purposefully deploy organisms in potentially polluted aquatic systems as biosensors (88, 89).

In the terrestrial environment, where heavy metal contamination and pesticide or herbicide accumulation can be critical problems, common soil organisms such as invertebrates (90) are useful Hsp-biomonitors of toxicants. For example, centipedes (*Lithobius*) collected from near a smelter had higher Hsp70 levels than those collected from unpolluted areas (91). Potentially, combinations of heavy metals can induce such distinctive patterns of Hsp expression in soil nematodes that these patterns can become diagnostic fingerprints for specific toxicants in soils (92).

Some aspects of the stress response, however, present problems for the use of Hsps as biomarkers in environmental toxicology. Because so many different stresses induce Hsps, investigators may be unable to attribute changes in Hsp expression to any particular environmental stress. Organisms in the field often undergo multiple stresses simultaneously, the interaction of which can yield significant Hsp expression even when no single monitored toxicant is at harmful levels. Conversely, Hsps induced by another stress can enhance tolerance of a toxicant whose presence is being monitored. Laboratory studies support the difficulty of teasing apart environmental factors and attributing Hsp induction to a single stressor. For example, freshwater sponges exhibit greater tolerance of pollutants following a sublethal heat stress (76). Among the vertebrates, diseased fish have elevated levels of Hsps in their tissues, and disease-related expression may interfere with the use of Hsps as a biomarker (93). Thus, because numerous factors can induce Hsp expression and stress tolerance, the utility of Hsps as biomarkers of environmental toxins may be limited.

#### ENVIRONMENTAL AND BIOLOGICAL CORRELATES OF THE HEAT-SHOCK RESPONSE AND HEAT-SHOCK PROTEINS

Many investigators view correlations of organismal traits (e.g., Hsp expression) and environmental or biological variables (e.g. level of environmental stress, developmental stage, distinctive role in a parasitic or symbiotic relationship with another species) as *prima facie* evidence of biological adaptation, and thus have actively sought such correlations in terms of the heat-shock response. While the probative value of such evidence in establishing adaptation has met with skepticism (94), in this section we consider the evidence for such correlations, whatever their meaning.

## VARIATION IN THE STRESS RESPONSE ALONG ENVIRONMENTAL GRADIENTS OF STRESS

To understand how Hsps result in stress tolerance at the organismal level, many investigators have characterized the stress response along gradients that occur in nature. One central question is whether organisms from environments with little stress have a different or reduced stress response compared with organisms from environments with much stress. Little and much stress might correspond to the center and edge of a species' range, low versus high elevation, xeric and hot versus mesic and cool climate, temperate versus tropical/polar latitude, low versus high intertidal, and so on. In general, the resulting data support a correlation among Hsp expression, stress tolerance, and gradients of environmental stress. These gradients have received uneven attention, however, and their study has yielded mixed results. Comparative studies across many degrees of latitude have not produced the same results as studies of gradients on smaller scales (e.g. diurnal or microclimatic variation in stress). Currently, not a single study has examined the stress response over the entire geographical distribution of a species; thus, whether species at the extremes of their distributions have an augmented heat-shock response is yet to be determined.

The majority of multi-species comparative studies focus on three aspects of the stress response: the minimum (threshold) and maximum temperatures at which Hsps are expressed and/or are present in cells, Hsp concentrations in cells, and the diversity of the specific Hsps that are expressed. Except for the work on threshold and maximum temperatures, much of this literature is a hodgepodge of disconnected studies that are seldom comparable because of methodological differences and permit few conclusions other than that species vary in the details of their stress response. Whether this variation has environmental correlates is uncertain. A rare and exemplary exception is the work of Bosch and colleagues on species of *Hydra* (95, 96); below we discuss this and other similar work.

In general, the threshold temperature for Hsp induction is correlated with the typical temperatures at which species live, with thermophilic species having a higher threshold than psychrophilic species. For example, a relatively coldwater, northern species of mussel (*Mytilus trossulus*) has a lower threshold for Hsp70 expression than its congener, *M. galloprovincialis*, a warm-water species with a more southern distribution (97). Limpet species that occur in the upper regions of the intertidal (*Lottia digitalis* and *Lottia pelta*) induce Hsps at 3-5[degree]C higher than the threshold for limpets that occur lower in the intertidal (*Tectura scutum*) (AL Haag & GE Hofmann, unpublished data). Subtidal species of the marine snail *Tegula* exhibit much the same pattern (98). Aggregate expression of Hsp70 family members (17) occurs at 3-4[degree]C higher in *Drosophila melanogaster* than in *D. ambigua*, a fruit fly of Palearctic origin (56). The same study reports a similar pattern for the desert ant *Cataglyphis* and *Formica polyctena*, a red wood ant from a temperate climate. One remarkable outcome of the *Cataglyphis* study is that Hsp synthesis in the desert ants continues at temperatures up to 45[degree]C, whereas temperatures above 39[degree]C inhibit Hsp synthesis in the temperate species. A similar pattern (although not as extreme) is evident for desert and non-desert *Drosophila* (22). These results suggest that translation itself may have an upper thermal maximum that varies among species adapted to different temperature environments.

Antarctic organisms represent a special case of psychrophily because the temperatures they experience are both extremely cold and extremely stable. In combination, do these conditions result in the evolutionary loss of a heat-shock response? In the subtidal alga, *Plocamium cartilagineum*, heat-inducible hsp70 and ubiquitin transcription still occur, although the threshold is a spectacularly cold 5[degree]C (99). Antarctic yeast species express Hsps at much lower temperatures than does *Saccharomyces*, and at least one species lacks inducible thermotolerance (100, 101). In Antarctic fish, the picture is not as clear. Although a broadly cross-reactive

anti-Hsp70 antibody can detect isoforms of Hsp70 in various tissues of the fish *Trematomus bernacchii*, heat shock temperatures from 6 to 10[degree]C do not induce additional Hsp70 accumulation (GE Hofmann, unpublished data). A member of the hsp70 gene family is present in two Antarctic fish species, *T. bernacchii* and *Notothenia coriiceps* (AC Whitmer & GE Hofmann, personal communication), and has been sequenced in Antarctic fish species (102). At the other extreme, some hyperthermophilic Archaea require temperatures in excess of 100[degree]C to induce Hsp expression (see Hsps of Archaea).

thermal stress gradients can be seasonal as well as geographic. In some cases, both Hsp expression and thermotolerance increase during warm seasons. The intertidal mussel *Mytilus californianus* displays significantly different Hsp induction profiles in summer than in winter, and summer-acclimatized mussels induce Hsps at a threshold temperature that is 6[degree]C higher than the threshold in winter-acclimatized mussels (103). However, whether the accentuated Hsp expression in mussels in summer results in greater thermotolerance at the organismal level is unknown. Fish (34, 35) and intertidal invertebrates (31) also vary seasonally in Hsp expression.

In addition to work we cite elsewhere, other studies examine geographical gradients in fish (104, 105, 105a), maize (106), reptiles (107), and *Drosophila* (108); intertidal gradients in limpets (27); diurnal temperature change in *Drosophila* (109) and intertidal mussels (29); diurnal variation in spruce trees (46); and seasonal variation in insects (110).

One issue for future consideration is whether Hsps in general are specialized to function at higher temperatures than other proteins (especially enzymes), and whether homologous Hsps of species from various thermal environments have corresponding variation in thermostability of function (111). For example, that an Hsp's resistance to thermal denaturation varies according to the thermal niche of the species that expresses it has been demonstrated for only a single Hsp, alpha-crystallin (112). Another issue is how differing thresholds of Hsp expression have evolved, whether through mutations in HSF, general thermostability of proteins and cells (113), or some other mechanism (114).

#### THE PARASITIC ENVIRONMENT

The roles of Hsps in host-parasite interactions have received considerable attention from both clinical and biological perspectives, with the majority of the research in two general categories. First, from the perspective of the host, Hsps expressed by invading parasites are potent antigens that elicit an immune response (115-117); parasites' Hsps are thus potentially useful in generating **vaccines** (118). From the perspective of the parasite, the synthesis of Hsps is a cellular defense mechanism that enables the parasite to live in different thermal environments throughout its life-cycle (119). Parasites that infect mammalian and avian hosts can undergo profound changes in temperature during the transition to these hosts (with internal temperatures of 37[degree]C or above) from ectotherm hosts or free-living stages. Induction of Hsps commonly accompanies this transition. Numerous studies have demonstrated developmentally regulated expression of Hsps in parasites; expression differs throughout the life-cycle both quantitatively and in the types of Hsps that temperature change induces. For example, mRNA transcripts for hsp70 and hsp83 homologues increase up to 100-fold as *Trypanosoma brucei* leaves the tsetse fly and enters a mammalian host (120). Aquatic snails release cercariae of the parasitic helminth, *Schistosoma mansoni*, into freshwater; cercariae penetrate human skin and develop into adult worms, eventually causing liver cirrhosis. The cercariae express two heat-inducible proteins that are not present in other stages (121).

Parasites that have an insect as the invertebrate vector have received much attention with regard to the developmentally regulated expression of Hsps. Examples include parasitic nematodes (122); cestode parasites (123); the malarial organism *Plasmodium* (124); *Borrelia burgdorferi*, the etiologic agent of Lyme disease (125); the protist *Leishmania* (126); *Trypanosoma cruzi* (127); and *Theileria* (128).

Some parasite life cycles do not involve an animal vector; a

free-living stage of the parasite occurs in water or soil and enters the host. In several cases, induction of Hsps accompanies the transition from the environment into the host. In the fungal parasite *Histoplasma capsulatum*, the temperature shift upon infection of a mammalian host cues both the transformation from a mycelial form to a budding yeast morphology and the expression of Hsps (129, 130). *Eimeria*, an intestinal parasite of numerous animals, expresses Hsp90 during infective life stages. *Eimeria* parasites are particularly interesting because this genus infects diverse hosts with correspondingly diverse body temperatures (e.g. marine fish, poultry, and cattle). However, specificity of infection is high at the species level, e.g. cattle are the exclusive host for *Eimeria bovis* (131). Whether the heat-shock response of *Eimeria* co-evolved with its speciation into these hosts is an open question.

Finally, the heat stress that infective life cycle stages of parasites experience is as diverse as their hosts. In nature, parasites of ectotherms can encounter dramatic shifts in temperature when their hosts' body temperature varies, as has been reported for parasites of reptiles, fish, and intertidal organisms (132, 133).

#### SYMBIOSIS

Just as Hsps may play an important role in parasitism, in which one species maintains a close but antagonistic relationship with others, they also function in symbiosis, in which interspecific relationships can be equally close but not adversarial. Perhaps the most general example of this point concerns mitochondria and chloroplasts, which evolved from endosymbionts that colonized other cells early in the history of life. These organelles often require proteins that are encoded in the nuclear genome and synthesized by the host cell, and hence must be imported into the organelle. Hsps play diverse roles in this process in mitochondria. A cytoplasmic Hsp70 family member maintains peptides in an unfolded conformation, which enables the peptides to pass through pores in the mitochondrial membrane; a mitochondrial Hsp70 is part of the protein machinery that imports the peptide; and the Hsp60/Hsp10 apparatus participates in the folding of the imported protein (134). Several groups of primitive eukaryotes contain still other endosymbiotically derived organelles, the hydrogenosome and the nucleomorph, whose Hsps share a characteristic sequence with those of mitochondria and proteobacteria (135-138). The Hsp sequence similarities have been used to suggest that hydrogenosomes may derive from mitochondria, share a common origin with mitochondria, or represent independent colonizations of early eukaryotic cells (135-137, 139).

Aside from endosymbiotically derived organelles, the best-studied symbioses concern bacterial endosymbionts that infect insects, including aphids, flies, ants, and cockroaches. Aphids, for example, harbor the bacterium *Buchnera* in specialized cells (bacteriocytes) within a distinctive structure in the body cavity, the bacteriome (140). The bacteria express a protein, symbionin, at especially high levels, and this protein is a member of the GroEL (Hsp60) family. Other bacterial chaperones, including GroES (Hsp10) and DnaK (Hsp70), are also present at high levels (140). A similar phenomenon is evident in tsetse flies (141).

The function and significance of these high Hsp levels is enigmatic. The Hsps apparently are not a response of the endosymbionts to a novel (and therefore stressful) host environment, as the *Buchnera*/aphid symbiosis is 150-250 million years old. The endosymbionts, however, have been evolving at an especially high rate; thus, the elevated molecular chaperones could be compensating for decreased protein stability due to the accumulation of numerous amino acid substitutions (142, 143). Nonetheless, the bacteria themselves can mount a strong heat-shock response when their host undergoes stress (144). Other relevant symbioses include X-bacteria in the symbiosomes of *Amoeba* (e.g. 145), *Bradyrhizobium* and *Rhizobium* in the root nodules of nitrogen-fixing plants (146), and the zooxanthellae component of corals (26, 30). A recurrent theme is that the endosymbionts modify the amount and/or diversity of Hsps present in the symbiosis. In some cases, this modification is thought to contribute to the maintenance of the

endosymbionts within the host, and in others to the augmentation of the heat-shock response of the symbiosis as a whole. Finally, *Wolbachia*, a bacterial endosymbiont that infects millions of arthropod species, both interferes with the mating of infected and uninfected hosts and can alter their constitutive expression of Hsp70 and Hsp90 family members (147). Simulated natural heat stress can diminish this reproductive interference, possibly by overriding the symbiont's effect on the host Hsps.

#### DEVELOPMENT

Many species exhibit characteristic and distinctive patterns of Hsp expression (or nonexpression) during the various stages of development, including gametogenesis, embryogenesis, and metamorphosis (e.g. 148-151). These patterns are often consistent with enhanced stress resistance in developmental stages that encounter unusual levels of environmental stress or during circumstances such as dormancy and diapause (see below). In other cases, developmental programs of Hsp expression ensue even in the absence of any obvious environmental stress. One common pattern is that one or more Hsps are not expressed in the initial phases of embryogenesis (152-156) or late in gametogenesis (157-161), possibly because Hsps can be harmful to developing cells (see Deleterious Aspects of Hsps). Parental provision of Hsps or hsp mRNAs can sometimes override gametic or embryonic absence of Hsp expression (162, 163); in other cases this absence presumably poses a significant problem for continued development in the face of stress (164). Stress not only can kill vulnerable developmental stages outright, but also can produce lasting damage to surviving organisms, such as the phenocopying of genetic defects; Hsps may minimize such defects (165, 166).

Adaptational analyses of the developmental expression of Hsps are diverse. Some plant seeds presumably must endure extremes of heat, desiccation, and other stresses before germinating, and some must germinate under especially challenging conditions. However, although seeds clearly undergo developmentally regulated expression of Hsps and embryos can express Hsps in response to environmental stress (167-169), few investigators have considered whether these patterns of expression are amplified or modified in species and ecotypes that naturally encounter especially challenging stress regimes (68, 170). Our state of knowledge is similar for fungal spores, which express particular Hsps in a developmentally regulated program (e.g. 171, 172). Several interesting case studies are available for animals, although a general pattern is yet to emerge. In the most spectacular example, encysted brine-shrimp (*Artemia*) embryos undergo developmental arrest, in which they may survive for years without environmental water or oxygen. The encysted embryos accumulate enormous concentrations (15[percent] of total protein) of a small Hsp (173-177) and trehalose (178), and suppress ubiquitination of damaged proteins (179). Non-adult *D. melanogaster* infest necrotic fruit, which can become extremely warm if it is sunlit (53, 54); this species mounts a massive heat-shock response, which is greatest in the developmental stages that presumably undergo the most exposure to natural heat stress (18, 19, 180). Other flies overwinter while at a particular developmental stage, and undergo considerable Hsp expression in response to cold (60-63). Later in development, ubiquitin may assist in the degeneration of flight muscles that are no longer needed after nuptial flights of insects (181). Finally, the temperature threshold for expression of Hsps may itself undergo modification; e.g. the threshold decreases in mammalian testis, in which gametogenesis normally occurs at lower temperatures than in the core of the body (182, 183).

#### AGING AND SENESCENCE

As mammals age, damage to proteins progressively accumulates, and both the ability to express Hsps (e.g. Hsp or hsp mRNA levels after a standard exposure to heat or other stress) and stress tolerance deteriorate (184, 185). Moreover, individual Hsps can become less able to mitigate the effects of stress on proteins as mammals age (186). In ecological and evolutionary terms, whether similar Hsp-aging relationships are important or even evident in wild organisms is unknown, although these relationships

occur in diverse species in the laboratory: *Drosophila* (187, 188), nematodes (189-191), and *Daphnia* (192).

These findings have provoked great interest in how Hsps potentially affect senescence and lifespan. A unifying hypothesis in the foregoing work is that protein damage, due primarily to oxidation/free radical activity, gradually accumulates during the life of a cell or organism and can lead to death if unabated; Hsps and other molecular stress responses ordinarily can mitigate this damage to some extent, and the decreasing expression of Hsps with age therefore contributes to mortality. If this hypothesis is correct, then **treatments** that both reduce damage to protein and increase Hsp expression (e.g. heat shock) should prolong life. In nematodes (*Caenorhabditis elegans*), some single-gene mutations that increase lifespan are associated with increased thermotolerance, but through as-yet-undescribed mechanisms (189-191). In *Drosophila*, heat shock extends lifespan (193), and this extension is enhanced in flies transformed with additional copies of the hsp70 gene (194). Nutrient deprivation can also extend life in rodents, presumably by reducing the metabolic rate and consequently, oxidative damage to proteins; starvation, however, variously increases, decreases, or has no effect on Hsp expression (195-199).

#### FITNESS CONSEQUENCES OF HSP EXPRESSION

##### GENERAL ISSUES AND BENEFICIAL ASPECTS OF HSPTS

Understanding the consequences of variation in Hsps and the stress response for Darwinian fitness requires a detailed appreciation of the mechanisms by which Hsps mitigate the impact of stress on individuals in natural populations. These mechanisms are becoming well understood at the level of model proteins with which Hsps can interact, but are progressively less well understood at the level of the cell, tissue, organ, and whole organism. At the level of the model protein, various stresses clearly either directly or indirectly result in conformational change, and Hsps typically promote the reacquisition or maintenance of the native structure and function by minimizing the tendency of non-native proteins to interact inappropriately (200, 201). In cells, stress-induced conformational change, protein aggregation, and molecular chaperoning of model proteins are also well established (200-202), and many cellular components differ in stress-tolerant and stress-intolerant cells (67).

Two primary issues impede the linkage of variation in these well-established mechanisms and phenomena to variation in the fitness of individual complex multicellular eukaryotes. First, is the variation in sensitivity to stress among cells, cell types, tissues, organs, and organisms attributable to a small number of critical lesions, especially sensitive targets of stress and functions of specific Hsps in protecting or repairing these lesions/targets? Or is variation in sensitivity to stress an aggregate function of a widespread and diverse impact of stress on cellular structures, with Hsps mitigating multiple lesions in diverse ways (21)? The former alternative may be more analytically tractable than the latter. Second, given that cells and organisms may have multiple Hsps in each Hsp family, multiple Hsp families, and multiple non-Hsp mechanisms of stress mitigation, how can we unambiguously establish the contribution or importance of any particular Hsp, Hsp family, or mechanism in the complex cell, tissue, organ, or organism? Much of the published literature on the functional consequences of Hsp expression for whole organisms or the cells they comprise runs afoul of these issues. Literally thousands of studies report correlations between Hsp expression, diverse biological functions in the face of stress, and stress tolerance, but these typically conclude that their findings are at best consistent with a role of one or more Hsps in stress tolerance. Evaluating the roles of single factors in complex systems is an ongoing challenge in most areas of the biological sciences, and the heat-shock field largely has not yet deployed counterparts of the solutions that other fields have developed.

One conspicuous and major exception includes techniques and approaches, primarily drawn from molecular biology and genetics, that allow the manipulation of individual Hsps or specific genes that encode them. In

rare instances, a species naturally may have an unusual genetic system (203) or a diminished suite of Hsps (95, 204) that accomplishes the same end; also, several chemical compounds may specifically inhibit one or more Hsps (e.g. 205, 206). The general implication of the resulting work (Table 1) is that, even in whole organisms or the cells they comprise, variation in single Hsps can be consequential for fitness. Some specific implications are as follows: Individual Hsps can have pleiotropic effects, interacting with multiple systems in diverse ways. Findings from manipulations of individual Hsps usually (but not always) are consistent with the outcome of correlative studies (see above). Finally, despite the huge body of work on Hsps and the growing use of manipulative techniques, we have remarkably little physiological insight into exactly how the activity of Hsps culminates in the enhanced stress tolerance of multicellular eukaryotes and the cells and tissues that they comprise.

Interestingly, one clear conclusion that correlative studies have yielded is that Hsps cannot account for the entirety of inducible stress tolerance (207-217). Indeed, some component of inducible stress tolerance may be unrelated to protein synthesis in general (214, 218, 219).

#### DELETERIOUS ASPECTS OF HSPS

The many advantages of the heat-shock response suggest that natural selection should maximize the expression of Hsps. By contrast, the genes encoding Hsps have not undergone unlimited amplification in the genome, and the Hsps themselves are subject to strict autoregulation by multiple molecular mechanisms (220). These contrary findings suggest that Hsps can have both positive and negative impacts on fitness, and that natural selection may have acted to balance these impacts in setting the level of Hsp expression. For example, while a small to moderate increases in Hsp70 levels enhance inducible thermotolerance in *Drosophila*, large increases in Hsp70 levels actually decrease thermotolerance (221); evolution thus may favor an intermediate level of Hsp70. A common theme in related work is that high levels of Hsps may be especially detrimental to cells or developmental stages in which cell growth and division proceed at high rates. *Drosophila* larvae transformed with extra copies of the hsp70 gene have greater larva-to-adult mortality and slower development than do control larvae; these strain differences are proportional to the number of Hsp-inducing heat shocks administered to the larvae (222). Larvae naturally varying in Hsp70 expression display a similar pattern (223). *Drosophila* cells engineered to express Hsp70 constitutively at first grow more slowly than control cells, but subsequently resume control growth rates once the Hsp70 is sequestered from the cytoplasm (164); indeed, *Drosophila* embryos remove Hsp70 from their cells rapidly after heat shock (224). A yeast strain that cannot express Hsp104 grows faster than its wild-type counterpart on some media (171). More generally, most animal species that have been studied do not mount a heat-shock response during early stages of embryogenesis (see Development), when protein synthesis may be especially intense.

These negative effects may have at least two nonexclusive explanations (222, 225, 226): First, Hsps at high concentration could be toxic, directly interfere with ongoing processes in the cell, or otherwise alter function to the detriment of the cell (220). Second, the synthesis and degradation of Hsps could consume an intolerably large fraction of a cell's or organism's nutrient and energy stores, and/or occupy so large a fraction of the synthetic/catabolic apparatus that the processing of other essential biomolecules suffers (226-228). Consistent with the first explanation, cellular sequestration of Hsp70 is correlated with the resumption of proliferation in cells constitutively expressing this protein (164). Also, overexpression of an Hsp70 family member inhibits protein secretion and reduction increases secretion in mammalian cells in culture (229-231); excess amounts of another Hsp70 family member can promote protein aggregation in vitro (M Borrelli & J Lepock, personal communication); and Hsp70 can perturb the normal structures of nascent polypeptides (232).

Tests of the second explanation have manipulated the costs of or resources for Hsp expression. Growth of corn in nitrogen-rich soil



increases the synthesis of Hsps in response to a standard heat shock (233); in plants grown in nitrogen-poor soil, other proteins may be catabolized to supply amino acids for synthesis of Hsps (234). These findings suggest that Hsp synthesis can be nitrogen-limited in plants. Starvation reduces the expression of Hsp 70 family members in mice (195). In *Drosophila* larvae, by contrast, co-expression of b-galactosidase and Hsps has no greater cost for growth and development than does expression of Hsps alone (225). Further study of this apparent trade-off of the benefits and disadvantages of Hsp expression, moreover, has the potential to link evolutionary and mechanistic views of this problem that heretofore have been separate (222, 225).

#### MICROEVOLUTIONARY VARIATION IN HSPS

Hsps are routinely touted as adaptations that arose and are maintained via natural selection for stress resistance. Origin and maintenance of a trait by selection require that it vary within populations, and that this intra-population variation have a genetic basis and affect the Darwinian fitness of individuals. Here we ask whether Hsps, the genes that encode them, and the factors that modify their expression display such patterns of variation and undergo stabilizing or directional selection in response to environmental stress.

First, not all intrapopulation and intraspecific variation results from genetic differences. For example, seasonal acclimatization and temperature acclimation in the laboratory can alter the minimum temperature at which *Gillichthys*, a gobiid fish, expresses an Hsp90 family member (34, 235). Seasonal acclimatization likewise affects Hsp70 levels in mussels (*Mytilus*) (103), and routine culture temperature affects the magnitude and temporal pattern of Hsp expression in HeLa cells (236). Such changes may stem from alterations in the cellular environment that modify the activation of HSF (113, 182, 236, 237). These changes, however, are not universal; laboratory thermal acclimation does not alter the thermal sensitivity of Hsp expression in fish hepatocytes in culture (238), *Drosophila* larvae (19), and mussels (103).

Even when acclimation and seasonal change are controlled, however, individuals within a population or species may vary in Hsp expression and/or the genes that determine it. Relevant research has examined this issue on two levels: direct sequence variation and restriction fragment length polymorphisms (RFLPs) (240). The sequence of hsp70 varies among strains of the parasite *Trypanosoma* (241, 242) and the nematode *C. elegans* (243), and among conspecifics for some but not all of the mammalian hsp70 family members (240), as does that of the 3' untranslated region of hsp27 in normotensive and hypertensive rats (244). RFLPs consistent with intraspecific variation either in the hsp genes or flanking regions are detectable in the hsp60 and hsp70 of the spirochete *Borrelia* (245), in multiple hsp70 family members of mammals (240, 246, 247), and in several plant species (248-250). One putative instance of intraspecific variation in hsp copy number concerns *D. melanogaster*, in which at least five nearly identical copies of hsp70 occur at two chromosomal loci. At locus 87A7, two copies are arranged as an inverted repeat (251, 252); at 87C1, two copies flank a region containing at least one additional copy (252, 253) plus numerous a/b repeats, which encode heat-inducible mRNAs of no proven function (254-257). Up to five additional hsp70 copies have been reported, with copy number varying among strains and time of year (253, 255, 258-261). However, these reports either cannot exclude that such variation is actually in intergenic regions or that the reports are for *Drosophila* cells in culture or mutagenized laboratory strains rather than wild or even wild-type strains. The organization of the two chromosomal loci reportedly varies among natural populations (253). A less equivocal instance of evolutionary change in gene copy number concerns *Arabidopsis*, in which ecotypes vary in the number of ubiquitin-encoding repeats (262). For all of the foregoing reports, the functional significance of intraspecific variation awaits elucidation or direct verification.

Hsp expression also exhibits genetic variation among individuals of a species; often, this variation is correlated with stress resistance (248,

250, 263-271). For example, isofemale lines of *Drosophila* founded from a single wild population differ more than twofold in Hsp70 expression; this variation is correlated with thermotolerance and is heritable (180, 223). Similarly, in the pathogenic fungus *Histoplasma*, naturally temperature-insensitive strains express more hsp70 mRNA and do so at lower temperatures than in a temperature-sensitive strain (272). In humans, fibroblasts isolated from desert-dwelling Turkmen express more Hsps and have greater thermotolerance than fibroblasts from residents of more equable climates (273); presumably, however, these peoples do not differ in body temperature.

Given that the patterns of variation necessary for natural selection occur within species, that selection can alter Hsp expression is not surprising. Laboratory evolution at high temperatures paradoxically lowers Hsp70 expression and inducible thermotolerance in *Drosophila* (19, 274), and selection for resistance to hyperthermic paralysis alters both the hsp68 promoter and the hsp70 locus in *Drosophila* (254, 275). Additional findings relevant to natural selection and its underlying genetic basis come from closely related species, some so similar that they hybridize. In the fish species *Poeciliopsis monacha* and *P. lucida*, an unusual genetic system permits the generation of hemiclinal lines in which the paternal genome varies against a constant maternal genome. Hemiclinal thermotolerance was most strongly related to Hsp70 and only secondarily to Hsp70 levels, in a pattern consistent with straightforward Mendelian inheritance of parental genotypes and adaptation to the local thermal environment. By contrast, the heat-shock response of interspecific hybrids of tomato (*Lycopersicon*) is not intermediate to the parental responses (276). Non-hybridizing congeners often exhibit a correlation among the actual or inferred incidence of thermal stress in their environment, heat-shock response, and stress tolerance. Such data are now available for diverse animals (see Variation in the Stress Response Along Environmental Gradients of Stress).

A particular problem with such species comparisons is that the interpretation of the observed patterns is readily confounded by phylogenetic and statistical issues. A more general problem with both laboratory evolution and species comparisons is that they describe only a supposed correlation of the heat-shock genes/proteins of interest with evolution and seldom can establish the importance of the genes/proteins of interest to evolutionary process and outcome (19, 20). Study of free-ranging organisms (277) with hsp transgenes (e.g. 109, 278) may contribute much to resolving these problems.

**THE EVOLUTIONARY HISTORY OF HSPTS AND THE GENES THAT ENCODE THEM**  
Hsps are among the most ancient and highly conserved of all proteins. Homologues of Hsps occur in every species in which they have been sought, and in all kingdoms of living things. Thus, Hsps represent a remarkable example of molecular "descent with modification" at the levels of gene sequence, genomic organization, regulation of gene expression, and protein structure and function. So clear are the patterns of descent and modifications that they can be used to establish the evolutionary origins and the phylogenetic affinities of the major groups of organisms.

#### HSPTS OF ARCHAEA AND EXCEPTIONAL PROKARYOTES

The Archaea or archaebacteria are the most extremophilic and most primitive organisms. The heat-shock response of extremophilic Archaea and nonarchaeal extremophiles occurs at remarkably high temperatures (279), e.g. 88[degree]C in *Sulfolobus* (279) and > 100[degree]C in the hyperthermophilic species designated ES4, a heterotrophic sulfur reducer isolated from a deep-sea hydrothermal vent (280). The archaeal genome encodes homologues of most Hsps represented in other prokaryotes and eukaryotes (279), as well as their consensus promoter sequences (281). Notably, the archaeal Hsp60 homologues assemble into a dual ring-like structure, termed a rosettasome or thermosome, that resembles the structure that the chaperones GroEL and GroES form in bacteria (279, 282, 283). The archaeal structures have ATPase activity and can bind denatured proteins (282). At least some Archaea and

Eubacteria differ in the number of monomers that comprise these structures (284). Surprisingly, the archaeal Hsp60s (e.g. TF55 of *Sulfolobus*) most closely resemble not a bacterial homologue, but the eukaryotic protein TCP1, which assembles into the t-complex polypeptide 1 ring complex (TRiC) in the cytosol (285, 286). Previously, Hsp60 homologues were thought to be absent from the eukaryotic cytosol. A growing body of evidence suggests that TCP1/TRiC and GroEL/GroES play comparable roles in their respective organisms and cellular compartments (287-289). Meanwhile, Trent (290) has suggested that the primary function of TF55 may be cytoskeletal, with molecular chaperoning a secondary or derived function.

Genes encoding Hsps are present even in the smallest of genomes. These include the genomes of mycoplasmas (291) and the nucleomorph, the vestigial nucleus of a phototrophic eukaryotic endosymbiont in cryptomonad algae (138). The section on symbiosis (see above) reviews the distribution of Hsps in various other organelles of endosymbiotic origin. Apparently, the problem of protein folding is ancient and ubiquitous, necessitating molecular chaperones in these diverse cases.

#### LARGE-SCALE EVOLUTION OF HSP GENES

The extraordinarily conserved nature of hsp genes (292) has facilitated their cloning, sequencing, and comparison in diverse organisms; their evolution is now becoming understood in detail. Gupta and colleagues have undertaken the most extensive surveys of hsp sequences, with a particular focus on organisms deemed critical to understanding the relationships of major taxa (292-297). The interpretations resulting from these comparisons relate to hypotheses about (a) the origin of eukaryotic cells, the eukaryotic nucleus, and endoplasmic reticulum (292, 296); (b) polyphyletic versus monophyletic origin of the major bacterial groups (292-294); and (c) the validity of the three-domain (Archaea, bacteria, and eukaryotes) dogma (292, 294). Whereas these interpretations are controversial, if not revolutionary, and therefore have not received universal acceptance, they nonetheless clearly illustrate how comparative analyses of hsp genes may address fundamental issues in evolutionary history.

On a less grand scale, hsp gene families represent superb case studies of how one or a small number of primitive genes can diversify to encode a suite of compartment- and function-specific proteins. One of many examples is dnaK, a single gene in Archaea and bacteria that has become the complex multigene hsp70 families of *Saccharomyces* (298), *Drosophila* (253, 299), and *Homo* (300). Another example concerns the small Hsps, which evolution recruited to become a major component of the lens of the eye: alpha crystallin (301). A growing body of work examines the discrete evolutionary events by which these changes may have occurred, including gene duplication/conversion events (302), retrotransposition, horizontal exchange of genomes, and others. New technologies promise to advance this work exponentially.

#### DISCRETE EXAMINATIONS OF MOLECULAR EVOLUTION

Ideally, a complete study of the evolutionary physiology of Hsps might examine how the following co-evolve as populations or how closely related species enter environments in which they face novel stresses: sequence (both coding and non-coding) of the gene(s) for a particular Hsp, regulation of hsp gene expression, the role and importance of the Hsp in stress resistance, and the intensities and durations of stress that the populations and species actually face. Much of the evolutionary physiological investigation of Hsps fails to attain this admittedly ambitious goal for one or more reasons: (a) The species under study are too distantly related to reconstruct the functional, environmental, and genetic events during their divergence; (b) molecular biology, manipulative genetics, physiology, and environmental assessment are not all possible for the species in question; or (c) the breadth of the techniques and approaches necessary to perform such research is too daunting for a single research program. Two case studies exemplify both how this goal could be approached and how far the field has yet to go to attain it.

The coelenterate *Hydra oligactis* and several of its congeners are the only multicellular eukaryotes reported not to express Hsps in response to heat shock and other stresses. Other congeners (e.g. *Hydra attenuata* and *H. magnipapillata*) have a well-developed stress response; these and other data for putative ancestors of *Hydra* suggest an evolutionary loss of Hsp expression in *H. oligactis* (95, 96). Physiologically and ecologically, *H. oligactis* is deficient in inducible stress tolerance and disappears from certain habitats in nature during periods of stress (95). Subsequent work suggests that, at least for Hsp70, the loss has occurred due to mutations that affect the stability of hsp70 mRNA, as *H. oligactis* has an hsp70 gene and expresses a heat-inducible hsp70 mRNA in quantities similar to that in the heat-tolerant *H. magnipapillata* (96).

Dipteran insects and their ancestors have undergone an evolutionary proliferation of hsp70 genes. Mosquitoes and *Drosophila* share a distinctly arranged duplication of the inducible hsp70 gene (303), suggesting that this proliferation predates the original diversification of the Diptera. Within the genus *Drosophila*, all groups other than the melanogaster subgroup of species apparently retain the primitive copy number of two (108, 304, 305). Within the melanogaster subgroup, all species examined to date have four copies except for *D. melanogaster* (253), which has at least five hsp70 copies in its haploid genome (see Microevolutionary Variation in Hsps). Curiously, *D. melanogaster* expresses no Hsp 100 family members, which are critical for thermotolerance in other organisms (204, 306). The proliferation of hsp70 copies is correlated with the ecological and biogeographic distribution of *Drosophila* species (19). Whereas most *Drosophila* species have small geographic ranges or narrow ecological niches, two of the melanogaster subgroup species (*simulans* and *melanogaster*) have cosmopolitan distributions, and a third (*yakuba*) is ecologically diverse throughout sub-Saharan Africa.

#### CONCLUSION

Ecological and evolutionary physiological analysis of heat-shock proteins may be nearing the end of its initial descriptive phase. Although accounts of spectacular levels of Hsp-mediated stress resistance and exceptional consequences of Hsp expression will continue to be newsworthy, the major patterns of Hsp expression in multicellular eukaryotes are becoming so obvious that additional descriptive work is becoming increasingly difficult to justify. Clearly, however, major questions remain unanswered. How the activities of Hsps at the molecular level culminate in organismal stress tolerance and how the hsp genes, their regulation, the function of the proteins they encode, and the environments faced by the organisms in which they occur all co-evolve are but two of the unresolved issues reviewed here. The perspective of evolutionary physiology can make significant contributions to the resolution of these and other issues. By placing results in actual environmental contexts, by assessing phenotypes of Hsps in the context of whole multicellular organisms, and by characterizing extant and historical variation in Hsps in natural populations and taxa, evolutionary physiologists can complement and extend a spectacular area of research that has been largely restricted to the molecular/cellular levels in the laboratory. By the same token, insights and techniques that laboratory-based investigators provide promise to continue to revolutionize the ecological and evolutionary study of Hsps. These approaches are both logical partners and necessary complements to one another (22, 307). Our understanding of Hsps has much to gain from the continued if not expanded synergy of these approaches.

Added material  
Martin E. Feder

Department of Organismal Biology and Anatomy and Committee on  
Evolutionary Biology, University of Chicago, 1027 East 57th Street,  
Chicago, Illinois 60637; e-mail: m-feder@uchicago.edu

Gretchen E. Hofmann

Department of Biology, University of New Mexico, Albuquerque, NM  
87131; e-mail: ghofmann@unm.edu

#### ACKNOWLEDGMENTS

We thank BR Bettencourt, K Chavez, UT D'OBrador, AP Nguyen, J Meredith-Patla, and AC Whitmer for bibliographic assistance and/or editorial advice, and Susan Lindquist for founding the bibliographic database that made this review possible. Research was supported by National Science Foundation grants 97-23298 and 97-23063. Finally and most importantly, we beg the indulgence of numerous investigators whom space limitations precluded us from citing directly here; their contributions have made the field what it is today. We urge all readers contemplating research in this area to scan the complete bibliography (13) available online at <http://www.AnnualReviews.org>.

Table 1 Phenotypes of multicellular eukaryotes, and the cells and tissues that they comprise, for which Hsps are necessary and/or sufficient(FNa)

#### FOOTNOTE

a In all cited work, specific Hsps have undergone experimental or natural manipulation.

#### LITERATURE CITED

1. Ritossa F. 1996. Discovery of the heat shock response. *Cell Stress Chaperones* 1:97-98
2. Lindquist S. 1986. The heat-shock response. *Annu. Rev. Biochem.* 55:1151-91
3. Gething MJ, Sambrook J. 1992. Protein folding in the cell. *Nature* 355:33-45
4. Morimoto RI, Tissieres A, Georgopoulos C, eds. 1994. *Heat Shock Proteins: Structure, Function and Regulation*. Cold Spring Harbor, NY: Cold Spring Harbor Lab. Press
5. Hartl FU. 1996. Molecular chaperones in cellular protein folding. *Nature* 381:571-80
6. Gething MJ, ed. 1997. *Guidebook to Molecular Chaperones and Protein-Folding Catalysts*. Oxford, UK: Oxford Univ. Press
7. Thomas PJ, Qu BH, Pedersen PL. 1995. Defective protein folding as a basis of human disease. *Trends Biochem. Sci.* 20:456-59
8. Boston RS, Viitanen PV, Vierling E. 1996. Molecular chaperones and protein folding in plants. *Plant Mol. Biol.* 32:191-222
9. Feige U, Morimoto RI, Yahara I, Polla BS, eds. 1996. *Stress-Inducible Cellular Responses*. Basel: Birkhauser
10. Feder ME, Parsell DA, Lindquist SL. 1995. The stress response and stress proteins. In *Cell Biology of Trauma*, ed. JJ Lemasters, C Oliver, pp. 177-91. Boca Raton, FL: CRC
11. Nover L. ed. 1991. *Heat Shock Response*. Boca Raton, FL: CRC
12. Somero GN. 1995. Proteins and temperature. *Annu. Rev. Physiol.* 57:43-68
13. Feder ME, Hofmann GE. 1998. Evolutionary and ecological physiology of heat-shock proteins and the heat-shock response: a comprehensive bibliography. <http://www.AnnualReviews.org>
14. Stephanou G, Alahiotis SN, Christodoulou C, Marmaras VJ. 1983. Adaptation of *Drosophila melanogaster* to temperature. Heat-shock proteins and survival in *Drosophila melanogaster*. *Dev. Genet.* 3:299-308
15. Alahiotis SN, Stephanou G. 1982. Temperature adaptation of *Drosophila* populations. The heat shock proteins system. *Comp. Biochem. Physiol.* 73B:529-33
16. Palter KB, Watanabe M, Stinson L, Mahowald AP, Craig EA. 1986. Expression and localization of *Drosophila melanogaster* hsp70 cognate proteins. *Mol. Cell Biol.* 6:1187-203
17. Hightower LE. 1995. Desert ants. *Science* 268:1417
18. Feder ME. 1998. Engineering candidate genes in studies of adaptation: the heat-shock protein Hsp70 in *Drosophila melanogaster*. *Am. Nat.* In press

19. Feder ME, Krebs RA. 1998. Natural and genetic engineering of thermotolerance in *Drosophila melanogaster*. *Am. Zool.* 38:503-17
20. Feder ME, Krebs RA. 1997. Ecological and evolutionary physiology of heat-shock proteins and the stress response in *Drosophila*: complementary insights from genetic engineering and natural variation. In *Stress, Adaptation, and Evolution*, ed. R Bijlsma, V Loeschcke, pp. 155-73. Basel: Birkhauser
21. Feder ME. 1996. Ecological and evolutionary physiology of stress proteins and the stress response: the *Drosophila melanogaster* model. In *Animals and Temperature: Phenotypic and Evolutionary Adaptation*, ed. IA Johnston, AF Bennett, pp. 79-102. Cambridge, UK: Cambridge Univ. Press
22. Huey RB, Bennett AF. 1990. Physiological adjustments to fluctuating thermal environments: an ecological and evolutionary perspective. In *Stress Proteins in Biology and Medicine*, ed. RI Morimoto, A Tissieres, C Georgopoulos, pp. 37-59. Cold Spring Harbor, NY: Cold Spring Harbor Lab. Press
23. Huey RB. 1991. Physiological consequences of habitat selection. *Am. Nat.* 137:S91-115
24. Bartholomew GA. 1964. The roles of physiology and behaviour in the maintenance of homeostasis in the desert environment. In *Homeostasis and Feedback Mechanisms*, ed. GM Hughes, pp. 7-29. Cambridge, UK: Cambridge Univ. Press
25. Sharp VA, Brown BE, Miller D. 1997. Heat shock protein (HSP 70) expression in the tropical reef coral *Goniopora djiboutiensis*. *J. Therm. Biol.* 22:11-19
26. Hayes RL, King CM. 1995. Induction of 70-kD heat shock protein in scleractinian corals by elevated temperature: significance for coral bleaching. *Mol. Mar. Biol. Biotechnol.* 4:36-42
27. Sanders BM, Hope C, Pascoe VM, Martin LS. 1991. Characterization of the stress protein response in two species of *Collisella* limpets with different temperature tolerances. *Physiol. Zool.* 64:1471-89
28. Sanders BM, Pascoe VM, Nakagawa PA, Martin LS. 1992. Persistence of the heat-shock response over time in a common *Mytilus* mussel. *Mol. Mar. Biol. Biotechnol.* 1:147-54
29. Hofmann GE, Somero GN. 1996. Protein ubiquitination and stress protein synthesis in *Mytilus trossulus* occurs during recovery from tidal emersion. *Mol. Mar. Biol. Biotechnol.* 5:175-84
30. Sharp VA, Miller D, Bythell JC, Brown BE. 1994. Expression of low molecular weight HSP 70 related polypeptides from the symbiotic sea anemone *Anemonia viridis* Forskal in response to heat shock. *J. Exp. Mar. Biol. Ecol.* 179:179-93
31. Hofmann GE, Somero GN. 1995. Evidence for protein damage at environmental temperatures: seasonal changes in levels of ubiquitin conjugates and hsp70 in the intertidal mussel *Mytilus trossulus*. *J. Exp. Biol.* 198:1509-18
32. Miller D, McLennan AG. 1988. The heat shock response of the cryptobiotic brine shrimp *Artemia*. I. A comparison of the thermotolerance of cysts and larvae. *J. Therm. Biol.* 13:119-24
33. Miller D, McLennan AG. 1988. The heat shock response of the cryptobiotic brine shrimp *Artemia*. II. Heat shock proteins. *J. Therm. Biol.* 13:125-34
34. Dietz TJ, Somero GN. 1992. The threshold induction temperature of the 90-kDa heat shock protein is subject to acclimatization in eurythermal goby fishes (genus *Gillichthys*). *Proc. Natl. Acad. Sci. USA* 89:3389-93
35. Feder SC, Yu Z, Spotila JR. 1994. Seasonal variation in heat shock proteins (hsp70) in stream fish under natural conditions. *J. Therm. Biol.* 19:335-41
36. Near JC, Easton DP, Rutledge PS, Dickinson DP, Spotila JS. 1990. Heat shock protein 70 gene expression in intact salamanders *Eurycea bislineata* in response to calibrated heat shocks and to high temperatures encountered in the field. *J. Exp. Zool.* 256:303-14
37. Gates DM. 1980. *Biophysical Ecology*. New York: Springer-Verlag
38. Kee SC, Nobel PS. 1986. Concomitant changes in high-temperature tolerance and heat-shock proteins in desert succulents. *Plant Physiol.* 80:596-98

39. Nagao RT, Kimpel JA, Key JL. 1990. Molecular and cellular biology of the heat-shock response. *Adv. Genet.* 28:235-74
40. Nguyen HT, Joshi CP, Klueva N, Weng J, Hendershot KL, Blum A. 1994. The heat-shock response and expression of heat-shock proteins in wheat under diurnal heat stress and field conditions. *Aust. J. Plant Physiol.* 21:857-67
41. Hamilton EW, Heckathorn SA, Downs CA, Schwarz TE, Coleman JS, Hallberg RL. 1996. Heat shock proteins are produced by field-grown naturally occurring plants in the summer in the temperate northeast U.S. *Bull. Ecol. Soc. Am.* 77, Suppl. Part 2:180 (Abstr.)
42. Kimpel JA, Key JL. 1985. Presence of heat shock mRNAs in field grown soybeans. *Plant Physiol.* 79:672-78
43. Hernandez LD, Vierling E. 1993. Expression of low molecular weight heat-shock proteins under field conditions. *Plant Physiol.* 101:1209-16
44. Hendershot KL, Weng J, Nguyen HT. 1992. Induction temperature of heat-shock protein synthesis in wheat. *Crop Sci.* 32:256-61
45. Burke JJ, Hatfield JL, Klein RP, Mullet JE. 1985. Accumulation of heat shock proteins in field-grown cotton. *Plant Physiol.* 78:394-98
46. Colombo SJ, Timmer VR, Colclough ML, Blumwald E. 1995. Diurnal variation in heat tolerance and heat shock protein expression in black spruce (*Picea mariana*). *Can. J. Forest Res.* 25:369-75
47. Morris GJ, Coulson G, Meyer MA, McLellan MR, Fuller BJ, et al. 1983. Cold shock--a widespread cellular reaction. *Cryo-Letters* 4:179-92
48. Danyluk J, Rassart E, Sarhan F. 1991. Gene expression during cold and heat shock in wheat. *Biochem. Cell Biol.* 69:383-91
49. Krishna P, Sacco M, Cherutti JF, Hill S. 1995. Cold-induced accumulation of hsp90 transcripts in *Brassica napus*. *Plant Physiol.* 107:915-23
50. Neven LG, Haskell DW, Guy CL, Denslow N, Klein PA, et al. 1992. Association of 70-kilodalton heat-shock cognate proteins with acclimation to cold. *Plant Physiol.* 99:1362-69
51. Van Berkel J, Salamini F, Gebhardt C. 1994. Transcripts accumulating during cold storage of potato (*Solanum tuberosum* L.) tubers are sequence related to stress-responsive genes. *Plant Physiol.* 104:445-52
52. Layne JR. 1991. Microclimate variability and the eurythermic natural of goldenrod gall fly (*Eurosta solidaginis*) larvae (Diptera: Tephritidae). *Can. J. Zool.* 69:614-17
53. Feder ME, Blair N, Figueras H. 1997. Natural thermal stress and heat-shock protein expression in *Drosophila* larvae and pupae. *Funct. Ecol.* 11:90-100
54. Feder ME. 1997. Necrotic fruit: a novel model system for thermal ecologists. *J. Therm. Biol.* 22:1-9
55. Wehner R, Marsh AC, Wehner S. 1992. Desert ants on a thermal tightrope. *Nature* 357:586-87
56. Gehring WJ, Wehner R. 1995. Heat shock protein synthesis and thermotolerance in *Cataglyphis*, an ant from the Sahara desert. *Proc. Natl. Acad. Sci. USA* 92:2994-98
57. Locke M, Noble EG. 1995. Stress proteins: the exercise response. *Can. J. Appl. Physiol.* 20:155-67
58. Brown IR, Rush SJ. 1996. In vivo activation of neural heat shock transcription factor HSF1 by a physiologically relevant increase in body temperature. *J. Neurosci. Res.* 44:52-57
59. Di YP, Repasky EA, Subjeck JR. 1997. Distribution of HSP70, protein kinase C, and spectrin is altered in lymphocytes during a fever-like hyperthermia exposure. *J. Cell. Physiol.* 172:44-54
60. Joplin KH, Denlinger DL. 1990. Developmental and tissue specific control of the heat shock induced 70 kDa related proteins in the flesh fly, *Sarcophaga crassipalpis*. *J. Insect Physiol.* 36:239-49
61. Joplin KH, Yocum GD, Denlinger DL. 1990. Cold shock elicits expression of heat shock proteins in the flesh fly *Sarcophaga crassipalpis*. *J. Insect Physiol.* 36:825-34
62. Yocum GD, Joplin KH, Denlinger DL. 1991. Expression of heat shock proteins in response to high and low temperature extremes in diapausing pharate larvae of the gypsy moth *Lymantria dispar*. *Arch. Insect Biochem. Physiol.* 18:239-50

63. Lee RE, Dommel RA, Joplin KH, Denlinger DL. 1995. Cryobiology of the freeze-tolerant gall fly *Eurosta solidaginis*: overwintering energetics and heat shock proteins. *Climate Res.* 5:61-67
64. Denlinger DL, Lee RE, Yocum GD, Kukal O. 1992. Role of chilling in the acquisition of cold tolerance and the capacitation to express stress proteins in diapausing pharate larvae of the gypsy moth *Lymantria dispar*. *Arch. Insect Biochem. Physiol.* 21:271-80
65. Matz JM, LaVoi KP, Moen RJ, Blake MJ. 1996. Cold-induced heat shock protein expression in rat aorta and brown adipose tissue. *Physiol. Behav.* 60:1369-74
66. Sills NS, Gorham DA, Carey HV. 1998. Stress protein expression in a mammalian hibernator. *FASEB J.* 12:A379
67. Li GC, Nussenzweig A. 1996. Thermotolerance and heat shock proteins: possible involvement of Ku autoantigen in regulating Hsp70 expression. See Ref. 9, pp. 121-37
68. Alamillo J, Almoguera C, Bartels D, Jordano J. 1995. Constitutive expression of small heat shock proteins in vegetative tissues of the resurrection plant *Craterostigma plantagineum*. *Plant Mol. Biol.* 29:1093-99
69. Pareek A, Singla SL, Kush AK, Grover A. 1997. Distribution patterns of HSP 90 protein in rice. *Plant Sci.* 125:221-30
70. Ma E, Haddad GG. 1997. Anoxia regulates gene expression in the central nervous system of *Drosophila melanogaster*. *Brain Res. Mol. Brain Res.* 46:325-28
71. Kultz D. 1996. Plasticity and stressor specificity of osmotic and heat shock responses of *Gillichthys mirabilis* gill cells. *Am. J. Physiol.* 271:C1181-93
72. de Pomerai D. 1996. Heat-shock proteins as biomarkers of pollution. *Hum. Exp. Toxicol.* 15:279-85
73. Sanders BM, Dyer SD. 1994. Cellular stress response. *Environ. Toxicol. Chem.* 13:1209-10
74. Sanders BM. 1993. Stress proteins in aquatic organisms: an environmental perspective. *Crit. Rev. Toxicol.* 23:49-75
75. Ryan JA, Hightower LE. 1996. Stress proteins as molecular biomarkers for environmental toxicology. See Ref. 9, pp. 411-24
76. Mueller WEG, Koziol C, Kurelec B, Dapper J, Batel R, Rinkevich B. 1995. Combinatory effects of temperature stress and nonionic organic pollutants on stress protein (hsp70) gene expression in the freshwater sponge *Ephydatia fluviatilis*. *Environ. Toxicol. Chem.* 14:1203-8
77. Cochrane BJ, Irby RB, Snell TW. 1991. Effects of copper and tributyltin on stress protein abundance in the rotifer *Brachionus plicatilis*. *Comp. Biochem. Physiol.* 98C:385-90
78. Krasko A, Scheffer U, Koziol C, Pancer Z, Batel R, et al. 1997. Diagnosis of sublethal stress in the marine sponge *Geodia cydonium*: application of the 70 kDa heat-shock protein and a novel biomarker, the Rab GDP dissociation inhibitor, as probes. *Aquat. Toxicol.* 37:157-68
79. Werner I, Nagel R. 1997. Stress proteins HSP60 and HSP70 in 3 species of amphipods exposed to cadmium, diazinon, dieldrin and fluoranthene. *Environ. Toxicol. Chem.* 16:2393-403
80. Ruffin P, Demuynck S, Hilbert JL, Dhainaut A. 1994. Stress protein in the polychaete annelid *Nereis diversicolor* induced by heat shock or cadmium exposure. *Biochimie* 76:423-27
81. Steinert SA, Pickwell GV. 1988. Expression of heat shock proteins and metallothionein in mussels exposed to heat stress and metal ion challenge. *Mar. Environ. Res.* 24:211-14
82. Veldhuizen Tsoerkan MB, Holwerda DA, van der Mast CA, Zandee DI. 1991. Synthesis of stress proteins under normal and heat shock conditions in gill tissue of sea mussels (*Mytilus edulis*) after chronic exposure to cadmium. *Comp. Biochem. Physiol.* 100C:699-706
83. Nascimento IA, Dickson KL, Zimmerman EG. 1996. Heat shock protein response to thermal stress in the Asiatic clam, *Corbicula fluminea*, J. *Aquat. Ecosystem Health* 5:231-38
84. Sanders BM, Martin LS, Howe SR, Nelson WG, Hegre ES, Phelps DK. 1994. Tissue-specific differences in accumulation of stress proteins in *Mytilus edulis* exposed to a range of copper concentrations. *Toxicol. Appl. Pharmacol.* 125:206-13



85. Ryan JA, Hightower LE. 1994. Evaluation of heavy-metal ion toxicity in fish cells using a combined stress protein and cytotoxicity assay. *Environ. Toxicol. Chem.* 13:1231-40
86. Dyer SD, Brooks GL, Dickson KL, Sanders BM, Zimmerman EG. 1993. Synthesis and accumulation of stress proteins in tissues of arsenite-exposed fathead minnows *Pimephales promelas*. *Environ. Toxicol. Chem.* 12:913-24
87. Vijayan MM, Pereira C, Forsyth RB, Kennedy CJ, Iwama GK. 1997. Handling stress does not affect the expression of hepatic heat shock protein 70 and conjugation enzymes in rainbow trout **treated** with beta-naphthoflavone. *Life Sci.* 61:117-27
88. Van Dyk TK, Majarian WR, Konstantinov KB, Young RM, Dhurjati PS, LaRossa RA. 1994. Rapid and sensitive pollutant detection by induction of heat shock gene-bioluminescence gene fusions. *Appl. Environ. Microbiol.* 60:1414-20
89. Veldhuizen Tsoerkan MB, Holwerda DA, de Bont AM, Smaal AC, Zandee DI. 1991. A field study on stress indices in the sea mussel, *Mytilus edulis*: application of the "stress approach" in biomonitoring. *Arch. Environ. Contam. Toxicol.* 21:497-504
90. Kohler HR, Triebkorn R, Stocker W, Kloetzel PM, Alberti G. 1992. The 70 kD heat shock protein (hsp 70) in soil invertebrates: a possible tool for monitoring environmental toxicants. *Arch. Environ. Contam. Toxicol.* 22:334-38
91. Pyza E, Mak P, Kramarz P, Laskowski R. 1997. Heat-shock proteins (Hsp70) as biomarkers in ecotoxicological studies. *Ecotoxicol. Environ. Safety* 38:244-51
92. Stringham EG, Candido EPM. 1994. Transgenic hsp16-lacZ strains of the soil nematode *Caenorhabditis elegans* as biological monitors of environmental stress. *Environ. Toxicol. Chem.* 13:1211-20
93. Forsyth RB, Candido EPM, Babich SL, Iwama GK. 1997. Stress protein expression in coho salmon with bacterial kidney disease. *J. Aquat. Anim. Health* 9:18-25
94. Rose MR, Lauder GV, eds. 1996. *Adaptation*. New York/London: Academic
95. Bosch TC, Krylow SM, Bode HR, Steele RE. 1988. Thermotolerance and synthesis of heat shock proteins: These responses are present in *Hydra attenuata* but absent in *Hydra oligactis*. *Proc. Natl. Acad. Sci. USA* 85:7927-31
96. Gellner K, Praetzel G, Bosch TC. 1992. Cloning and expression of a heatinducible hsp70 gene in two species of *Hydra* which differ in their stress response. *Eur. J. Biochem.* 210:683-91
97. Hofmann GE, Somero GN. 1996. Interspecific variation in thermal denaturation of proteins in the congeneric mussels *Mytilus trossulus* and *M. galloprovincialis*: evidence from the heatshock response and protein ubiquitination. *Mar. Biol.* 126:65-75
98. Tomanek L, Somero GN. 1997. The effect of temperature on protein synthesis in snails of the genus *Tegula* from the sub- and intertidal zone. *Am. Zool.* 37:188A
99. Vayda ME, Yuan ML. 1994. The heat shock response of an Antarctic alga is evidence at 5 degrees C. *Plant Mol. Biol.* 24:229-33
100. Berg GR, Inniss WE, Heikkila JJ. 1987. Stress proteins and thermotolerance in psychrotrophic yeasts from Arctic environments. *Can. J. Microbiol.* 33:383-89
101. Deegenaars ML, Watson K. 1997. Stress proteins and stress tolerance in an Antarctic, psychrophilic yeast, *Candida psychrophila*. *FEMS Microbiol. Lett.* 151:191-96
102. Carratu L, Maresca B. 1997. Evolutionary adaptation of hsp70 gene in Antarctic fish. *Exp. Biol. Online* 2:C5.1 (Abstr.)
103. Roberts DA, Hofmann GE, Somero GN. 1997. Heat-shock protein expression in *Mytilus californianus*: acclimatization (seasonal and tidal-height comparisons) and acclimation effects. *Biol. Bull.* 192:309-20
104. Norris CE, diIorio PJ, Schultz RJ, Hightower LE. 1995. Variation in heat shock proteins within tropical and desert species of poeciliid fishes. *Mol. Biol. Evol.* 12:1048-62
105. White CN, Hightower LE, Schultz RJ. 1994. Variation in heat-shock

- proteins among species of desert fishes (Poeciliidae, Poeciliopsis). Mol. Biol. Evol. 11:106-19
- 105a. Maresca B, Patriarcha E, Goldenberg C, Sacco M. 1988. Heat shock and cold adaptation in Antarctic fishes: a molecular approach. Comp. Biochem. Physiol. 90B:623-29
106. Ristic Z, Williams G, Yang G, Martin B, Fullerton S. 1996. Dehydration, damage to cellular membranes, and heat-shock proteins in maize hybrids from different climates. J. Plant Physiol. 149:424-32
107. Ulmasov KA, Shammakov S, Karaev K, Evgenev MB. 1992. Heat shock proteins and thermoresistance in lizards. Proc. Natl. Acad. Sci. USA 89:1666-70
108. Konstantopoulou I, Drosopoulou E, Scouras ZG. 1997. Variations in the heat-induced protein pattern of several *Drosophila montium* subgroup species (Diptera: Drosophilidae). Genome 40:132-37
109. Feder ME, Cartano NV, Milos L, Krebs RA, Lindquist SL. 1996. Effect of engineering Hsp70 copy number on Hsp70 expression and tolerance of ecologically relevant heat shock in larvae and pupae of *Drosophila melanogaster*. J. Exp. Biol. 199:1837-44
110. Nath BB, Lakhotia SC. 1989. Heat-shock response in a tropical Chironomus: seasonal variation in response and the effect of developmental stage and tissue type on heat shock protein synthesis. Genome 32:676-86
111. Hofmann GE. 1996. Molecular chaperone activity of the stress protein Hsc70 purified from an eurythermal goby, *Gillichthys mirabilis*. Am. Zool. 36:36A
112. McFall-Ngai M, Horwitz J. 1990. A comparative study of the thermal stability of the vertebrate eye lens: Antarctic fish to the desert iguana. Exp. Eye Res. 50:703-9
113. Clos J, Rabindran S, Wisniewski J, Wu C. 1993. Induction temperature of human heat shock factor is reprogrammed in a *Drosophila* cell environment. Nature 364:252-55
114. Carratu L, Franceschelli S, Pardini CL, Kobayashi GS, Horvath I, et al. 1996. Membrane lipid perturbation modifies the set point of the temperature of heat shock response in yeast. Proc. Natl. Acad. Sci. USA 93:3870-75
115. Maresca B, Kobayashi GS. 1994. Hsp70 in parasites: as an inducible protective protein and as an antigen. Experientia 50:1067-74
116. Kaufmann SH. 1992. The cellular immune response to heat shock proteins. Experientia 48:640-43
117. Polla BS. 1991. Heat shock proteins in host-parasite interactions. Immunol. Today 12:A38-41
118. Newport GR. 1991. Heat shock proteins as **vaccine** candidates. Semin. Immunol. 3:17-24
119. Tsuji N, Ohta M, Fujisaki K. 1997. Expression of a 70-kDa heat-shock-related protein during transformation from freeliving infective larvae to the parasitic stage in *Strongyloides venezuelensis*. Parasitol. Res. 83:99-102
120. Van der Ploeg LH, Giannini SH, Cantor CR. 1985. Heat shock genes: regulatory role for differentiation in parasitic protozoa. Science 228:1443-46
121. Neumann S, Ziv E, Lantner F, Schechter I. 1993. Regulation of HSP70 gene expression during the life cycle of the parasitic helminth *Schistosoma mansoni*. Eur. J. Biochem. 212:589-96
122. van Leeuwen MA. 1995. Heat-shock and stress response of the parasitic nematode *Haemonchus contortus*. Parasitol. Res. 81:706-9
123. Ernani FP, Teale JM. 1993. Release of stress proteins from *Mesocostoides corti* is a brefeldin A-inhibitable process: evidence for active export of stress proteins. Infect. Immun. 61:2596-601
124. Syin C, Goldman ND. 1996. Cloning of a *Plasmodium falciparum* gene related to the human 60-kDa heat shock protein. Mol. Biochem. Parasitol. 79:13-19
125. Cluss RG, Boothby JT. 1990. Thermoregulation of protein synthesis in *Borrelia burgdorferi*. Infect. Immun. 58:1038-42
126. Hubel A, Krobitch S, Horauf A, Clos J. 1997. The *Leishmania* major Hsp 100 is required chiefly in the mammalian stage of the parasite. Mol. Cell. Biol. 17:5987-95

127. Giambiagi-de Marval M, Souto-Padron T, Rondinelli E. 1996. Characterization and cellular distribution of heat-shock proteins HSP70 and HSP60 in *Trypanosoma cruzi*. *Exp. Parasitol.* 83:335-45
128. Daubenberger C, Heussler V, Gobright E, Wijngaard P, Clevers HC, et al. 1997. Molecular characterisation of a cognate 70 kDa heat shock protein of the protozoan *Theileria parva*. *Mol. Biochem. Parasitol.* 85:265-69
129. Shearer GJ, Birge CH, Yuckenberg PD, Kobayashi GS, Medoff G. 1987. Heat-shock proteins induced during the mycelial-to-yeast transitions of strains of *Histoplasma capsulatum*. *J. Gen. Microbiol.* 133:3375-82
130. Maresca B. 1995. Unraveling the secrets of *Histoplasma capsulatum*. A model to study morphogenic adaptation during parasite host/host interaction. *Verh. K. Acad. Geneesk. Belg.* 57:133-56
131. Clark TG, Abrahamsen MS, White MW. 1996. Developmental expression of heat shock protein 90 in *Eimeria bovis*. *Mol. Biochem. Parasitol.* 78:259-63
132. Tirard CT, Grossfeld RM, Volety AK, Chu FLE. 1995. Heat shock proteins of the oyster parasite *Perkinsus marinus*. *Dis. Aquat. Organisms* 22:147-51
133. Ulmasov KA, Ovezmukhammedov A, Karaev KK, Evgenov MB. 1988. Molecular mechanisms of adaptation to hyperthermia in higher organisms. III. Induction of heat-shock proteins in two *Leishmania* species. *Mol. Biol.* 22:1583-89
134. Ryan MT, Naylor DJ, Hoj PB, Clark MS, Hoogenraad NJ. 1997. The role of molecular chaperones in mitochondrial protein import and folding. *Int. Rev. Cytol.* 174:127-93
135. Germot A, Philippe H, Le Guyader H. 1997. Evidence for loss of mitochondria in microsporidia from a mitochondrial-type HSP70 in *Nosema locustae*. *Mol. Biochem. Parasitol.* 87:159-68
136. Bui ET, Bradley PJ, Johnson PJ. 1996. A common evolutionary origin for mitochondria and hydrogenosomes. *Proc. Natl. Acad. Sci. USA* 93:9651-56
137. Sogin ML. 1997. Organelle origins: energy-producing symbionts in early eukaryotes? *Curr. Biol.* 7:R315-17
138. Hofmann CJ, Rensing SA, Hauber MM, Martin WF, Muller SB, et al. 1994. The smallest known eukaryotic genomes encode a protein gene: towards an understanding of nucleomorph functions. *Mol. Gen. Genet.* 243:600-4
139. Germot A, Philippe H, Le Guyader H. 1996. Presence of a mitochondrial-type 70-kDa heat shock protein in *Trichomonas vaginalis* suggests a very early mitochondrial endosymbiosis in eukaryotes. *Proc. Natl. Acad. Sci. USA* 93:14614-17
140. Baumann P, Moran NA, Baumann L. 1997. The evolution and genetics of aphid endosymbionts. *BioScience* 47:12-20
141. Aksoy S. 1995. Molecular analysis of the endosymbionts of tsetse flies: 16S rDNA locus and over-expression of a chaperonin. *Insect Mol. Biol.* 4:23-29
142. Moran NA, Von Dohlen CD, Baumann P. 1995. Faster evolutionary rates in endosymbiotic bacteria than in cospeciating insect hosts. *J. Mol. Evol.* 41:727-31
143. Moran NA. 1996. Accelerated evolution and Muller's ratchet in endosymbiotic bacteria. *Proc. Natl. Acad. Sci. USA* 93:2873-78
144. Morioka M, Ishikawa H. 1992. Mutualism based on stress: selective synthesis and phosphorylation of a stress protein by an intracellular symbiont. *J. Biochem.* 111:431-35
145. Hong HK, Choi JY, Ahn TI. 1994. Molecular biological studies on the heat-shock responses in *Amoeba proteus*: I. Detection of heat-shock proteins. *Korean J. Zool.* 37:554-64
146. Choi EY, Ahn GS, Jeon KW. 1991. Elevated levels of stress proteins associated with bacterial symbiosis in *Amoeba proteus* and soybean root nodule cells. *Biosystems* 25:205-12
147. Feder ME, Karr TL. 1997. Evolutionarily significant consequences of the heat shock response for *Drosophila* and its endosymbiont *Wolbachia*. *Am. Zool.* 37:8A
148. Dix DJ. 1997. Hsp70 expression and function during gametogenesis. *Cell Stress Chaperones* 2:73-77

149. Lin JC, Song CW. 1993. Heat shock gene expression and development. I. An overview of fungal, plant, and poikilothermic animal developmental systems. *Dev. Genet.* 14:1-5
150. Winter J, Sinibaldi R. 1991. The expression of heat shock protein and cognate genes during plant development. *Results Prob. Cell Differ.* 17:85-105
151. Mosser DD, Duchaine J, Bourget L, Martin LH. 1993. Heat shock gene expression and development. II. An overview of mammalian and avian developmental systems. *Dev. Genet.* 14:87-91
152. Heikkila JJ, Ohan N, Tam Y, Ali A. 1997. Heat shock protein gene expression during *Xenopus* development. *Cell. Mol. Life Sci.* 53:114-21
153. Muller WU, Li GC, Goldstein LS. 1985. Heat does not induce synthesis of heat shock proteins or thermotolerance in the earliest stage of mouse embryo development. *Int. J. Hypertherm.* 1:97-102
154. Edwards JL, Ealy AD, Monterroso VH, Hansen PJ. 1997. Ontogeny of temperature-regulated heat shock protein 70 synthesis in preimplantation bovine embryos. *Mol. Reprod. Dev.* 48:25-33
155. Dura JM. 1981. Stage dependent synthesis of heat shock induced proteins in early embryos of *Drosophila melanogaster*. *Mol. Gen. Genet.* 184:381-85
156. Welte MA, Tetrault JM, Dellavalle RP, Lindquist SL. 1993. A new method for manipulating transgenes: engineering heat tolerance in a complex, multicellular organism. *Curr. Biol.* 3:842-53
157. Gagliardi D, Breton C, Chaboud A, Vergne P, Dumas C. 1995. Expression of heat shock factor and heat shock protein 70 genes during maize pollen development. *Plant Mol. Biol.* 29:841-56
158. Hendrey J, Kola I. 1991. Thermolability of mouse oocytes is due to the lack of expression and/or inducibility of Hsp70. *Mol. Reprod. Dev.* 28:1-8
159. Curci A, Bevilacqua A, Mangia F. 1987. Lack of heat-shock response in preovulatory mouse oocytes. *Dev. Biol.* 123:154-60
160. Curci A, Bevilacqua A, Fiorenza MT, Mangia F. 1991. Developmental regulation of heat-shock response in mouse oogenesis: identification of differentially responsive oocyte classes during Graafian follicle development. *Dev. Biol.* 144:362-68
161. Zakeri ZF, Welch WJ, Wolgemuth DJ. 1990. Characterization and inducibility of hsp 70 proteins in the male mouse germ line. *J. Cell Biol.* 111:1785-92
162. Bedard PA, Brandhorst BP. 1986. Translational activation of maternal mRNA encoding the heat-shock protein hsp90 during sea urchin embryogenesis. *Dev. Biol.* 117:286-93
163. Gordon S, Bharadwaj S, Hnatov A, Ali A, Ovsenek N. 1997. Distinct stress-inducible and developmentally regulated heat shock transcription factors in *Xenopus* oocytes. *Dev. Biol.* 181:47-63
164. Feder JH, Rossi JM, Solomon J, Solomon N, Lindquist S. 1992. The consequences of expressing Hsp70 in *Drosophila* cells at normal temperatures. *Genes Dev.* 6:1402-13
165. Mitchell HK, Moller G, Petersen NS, Lipps-Sarmiento L. 1979. Specific protection from phenocopy induction by heat shock. *Dev. Genet.* 1:181-92
166. Welte MA, Duncan I, Lindquist S. 1995. The basis for a heat-induced developmental defect: defining crucial lesions. *Genes Dev.* 9:2240-50
167. Wehmeyer N, Hernandez LD, Finkelstein RR, Vierling E. 1996. Synthesis of small heat-shock proteins is part of the developmental program of late seed maturation. *Plant Physiol.* 112:747-57
168. Duck N, McCormick S, Winter J. 1989. Heat shock protein Hsp70 cognate gene expression in vegetative and reproductive organs of *Lycopersicon esculentum*. *Proc. Natl. Acad. Sci. USA* 86:3674-78
169. Coca MA, Almoguera C, Jordano J. 1994. Expression of sunflower low-molecular-weight heat-shock proteins during embryogenesis and persistence after germination: localization and possible functional implications. *Plant Mol. Biol.* 25:479-92
170. Helm KW, Petersen NS, Abernethy RH. 1989. Heat-shock response of germinating embryos of wheat: effects of imbibition time and seed vigor.

Plant Physiol. 90:598-605

171. Sanchez Y, Taulien J, Borkovich KA, Lindquist S. 1992. Hsp104 is required for tolerance to many forms of stress. EMBO J. 11:2357-64

172. Silva AM, Juliani MH, da Costa JJ, Bonato MC. 1987. Acquisition of thermotolerance during development of *Blastocladiella emersonii*. Biochem. Biophys. Res. Commun. 144:491-98

173. Liang P, Amons R, Clegg JS, MacRae TH. 1997. Molecular characterization of a small heat shock/alpha-crystallin protein in encysted *Artemia* embryos. J. Biol. Chem. 272:19051-58

174. Liang P, Amons R, Macrae TH, Clegg JS. 1997. Purification, structure and in vitro molecular-chaperone activity of *Artemia* p26, a small heat-shock/alpha-crystallin protein. Eur. J. Biochem. 243:225-32

175. Jackson SA, Clegg JS. 1996. Ontogeny of low molecular weight stress protein p26 during early development of the brine shrimp, *Artemia franciscana*. J. Exp. Biol. 200:467-75

176. Clegg JS, Jackson SA, Liang P, MacRae TH. 1995. Nuclear-cytoplasmic translocations of protein p26 during aerobic-anoxic transitions in embryos of *Artemia franciscana*. Exp. Cell Res. 219:1-7

177. Clegg JS, Jackson SA, Warner AH. 1994. Extensive intracellular translocations of a major protein accompany anoxia in embryos of *Artemia franciscana*. Exp. Cell Res. 212:77-83

178. Clegg JS, Jackson SA. 1992. Aerobic heat shock activates trehalose synthesis in embryos of *Artemia franciscana*. FEBS Lett. 303:45-47

179. Anchordoguy TJ, Hand SC. 1994. Acute blockage of the ubiquitin-mediated proteolytic pathway during invertebrate quiescence. Am. J. Physiol. 267:R895-900

180. Krebs RA, Feder ME, Lee J. 1998. Heritability of expression of the 70-kD heat-shock protein in *Drosophila melanogaster* and its relevance to the evolution of thermotolerance. Evolution 52:841-47

181. Davis WL, Jacoby BH, Goodman DB. 1994. Immunolocalization of ubiquitin in degenerating insect flight muscle. Histochem. J. 26:298-305

182. Sarge KD, Bray AE, Goodson ML. 1995. Altered stress response in testis. Nature 374:126

183. Sarge KD. 1995. Male germ cell-specific alteration in temperature set point of the cellular stress response. J. Biol. Chem. 270:18745-48

184. Holbrook NJ, Udelsman R. 1994. Heat shock protein gene expression in response to physiologic stress and aging. See Ref. 4, pp. 577-93

185. Lee YK, Manalo D, Liu AY. 1996. Heat shock response, heat shock transcription factor and cell aging. Biol. Signals 5:180-91

186. Shpund S, Gershon D. 1997. Alterations in the chaperone activity of HSP70 in aging organisms. Arch. Gerontol. Geriatr. 24:125-31

187. Wheeler JC, Bieschke ET, Tower J. 1995. Muscle-specific expression of *Drosophila* hsp70 in response to aging and oxidative stress. Proc. Natl. Acad. Sci. USA 92:10408-12

188. Marin R, Valet JP, Tanguay RM. 1993. Heat shock induces changes in the expression and binding of ubiquitin in senescent *Drosophila melanogaster*. Dev. Genet. 14:78-86

189. Lithgow GJ, White TM, Hinerfeld DA, Johnson TE. 1994. Thermotolerance of a long-lived mutant of *Caenorhabditis elegans*. J. Gerontol. 49B:270-76

190. Lithgow GJ, White TM, Melov S, Johnson TE. 1995. Thermotolerance and extended life-span conferred by single-gene mutations and induced by thermal stress. Proc. Natl. Acad. Sci. USA 92:7540-44

191. Lithgow GJ. 1996. Invertebrate gerontology: the age mutations of *Caenorhabditis elegans*. BioEssays 18:809-15

192. Bond JA, Gonzalez CRM, Bradley BP. 1993. Age-dependent expression of proteins in the cladoceran *Daphnia magna* under normal and heat-stress conditions. Comp. Biochem. Physiol. 106B:913-17

193. Khazaeli AA, Tatar M, Pletcher SD, Curtsinger JW. 1997. Heat-induced longevity extension in *Drosophila*. I. Heat treatment, **mortality**, and thermotolerance. J. Gerontol. 52A:B48-52

194. Tatar M, Khazaeli AA, Curtsinger JW. 1997. Chaperoning extended life. Nature 390:30

195. Dhahbi JM, Mote PL, Tillman JB, Walford RL, Spindler SR. 1997. Dietary energy tissue-specifically regulates endoplasmic reticulum

- chaperone gene expression in the liver of mice. *J. Nutr.* 127:1758-64
196. Heydari AR, Conrad CC, Richardson A. 1995. Expression of heat shock genes in hepatocytes is affected by age and food restriction in rats. *J. Nutr.* 125:410-18
197. Pahlavani MA, Harris MD, Moore SA, Richardson A. 1996. Expression of heat shock protein 70 in rat spleen lymphocytes is affected by age but not by food restriction. *J. Nutr.* 126:2069-75
198. Lu Q, Wallrath LL, Granok H, Elgin SC. 1993. Expression of heat shock protein 70 is altered by age and diet at the level of transcription. *Mol. Cell Biol.* 13:2909-18
199. Masoro EJ. 1996. Possible mechanisms underlying the antiaging actions of caloric restriction. *Toxicol. Pathol.* 24:738-41
200. Parsell DA, Lindquist S. 1993. The function of heat-shock proteins in stress tolerance: degradation and reactivation of damaged proteins. *Annu. Rev. Genet.* 27:437-96
201. Parsell DA, Lindquist S. 1994. Heat shock proteins and stress tolerance. See Ref. 9, pp. 457-94
202. Bensaude O, Bellier S, Dubois MF, Giannoni F, Nguyen VT. 1996. Heat-shock induced protein modifications and modulation of enzyme activities. See Ref. 9, pp. 199-219
203. diIorio PJ, Holsinger K, Schultz RJ, Hightower LE. 1996. Quantitative evidence that both Hsc70 and Hsp70 contribute to thermal adaptation in hybrids of the livebearing fishes *Poeciliopsis*. *Cell Stress Chaperones* 1:139-47
204. Feder ME, Lindquist SL. 1992. Evolutionary loss of a heat shock protein. *Am. Zool.* 32:51A (Abstr.)
205. Whitesell L, Cook P. 1996. Stable and specific binding of heat shock protein 90 by geldanamycin disrupts **glucocorticoid** receptor function in intact cells. *Mol. Endocrinol.* 10:705-12
206. Elia G, Santoro MG. 1994. Regulation of heat shock protein synthesis by quercetin in human erythroleukaemia cells. *Biochem. J.* 300:201-9
207. Bonham-Smith PC, Kapoor M, Bewley JD. 1987. Establishment of thermotolerance in maize by exposure to stresses other than a heat shock does not require heat shock protein synthesis. *Plant Physiol.* 85:575-80
208. Xiao CM, Mascarenhas JP. 1985. High temperature-induced thermotolerance in pollen tubes of *Tradescantia* and heat-shock proteins. *Plant Physiol.* 78:887-90
209. VanBogelen RA, Acton MA, Neidhardt FC. 1987. Induction of the heat shock regulon does not produce thermotolerance in *Escherichia coli*. *Genes Dev.* 1:525-31
210. Yocum GD, Denlinger DL. 1992. Prolonged thermotolerance in the flesh fly *Sarcophaga crassipalpis* does not require continuous expression or persistence of the 72 kDa heat-shock protein. *J. Insect Physiol.* 38:603-9
211. Boon-Niermeijer EK, Tuyl M, van de Scheur H. 1986. Evidence for two states of thermotolerance. *Int. J. Hypertherm.* 2:93-105
212. Smith BJ, Yaffe MP. 1991. Uncoupling thermotolerance from the induction of heat shock proteins. *Proc. Natl. Acad. Sci. USA* 88:11091-94
213. Easton DP, Rutledge PS, Spotila JR. 1987. Heat shock protein induction and induced thermal tolerance are independent in adult salamanders. *J. Exp. Zool.* 241:263-67
214. Dingley F, Maynard Smith J. 1968. Temperature acclimatization in the absence of protein synthesis of *Drosophila subobscura*. *J. Insect Physiol.* 14:1185-94
215. Finnell RH, Van Waes M, Bennett GD, Eberwine JH. 1993. Lack of concordance between heat shock proteins and the development of tolerance to teratogen-induced neural tube defects. *Dev. Genet.* 14:137-47
216. Fisher B, Kraft P, Hahn GM, Anderson RL. 1992. Thermotolerance in the absence of induced heat shock proteins in a murine lymphoma. *Cancer Res.* 52:2854-61
217. Watson K, Dunlop G, Cavicchioli R. 1984. Mitochondrial and cytoplasmic protein syntheses are not required for heat shock acquisition of ethanol and thermotolerance in yeast. *FEBS Lett.* 172:299-302
218. Widelitz RB, Magun BE, Gerner EW. 1986. Effects of cycloheximide on thermotolerance expression, heat shock protein synthesis, and heat shock

- protein mRNA accumulation in rat fibroblasts. *Mol. Cell Biol.* 6:1088-94
219. Jozwiak Z, Leyko W. 1992. Role of membrane components in thermal injury of cells and development of thermotolerance. *Int. J. Radiat. Biol.* 62:743-56
220. Lindquist S. 1993. Autoregulation of the heat-shock response. In *Translational Regulation of Gene Expression 2*, ed. J. Ilan, pp. 279-320. New York: Plenum
221. Krebs PA, Feder ME. 1998. Hsp70 and larval thermotolerance in *Drosophila melanogaster*: How much is enough and when is more too much? *J. Insect Physiol.* 44:1091-1101
222. Krebs PA, Feder ME. 1997. Deleterious consequences of Hsp70 overexpression in *Drosophila melanogaster* larvae. *Cell Stress Chaperones* 2:60-71
223. Krebs PA, Feder ME. 1997. Natural variation in the expression of the heat-shock protein Hsp70 in a population of *Drosophila melanogaster*, and its correlation with tolerance of ecologically relevant thermal stress. *Evolution* 51:173-79
224. Welte MA. 1994. Thermotolerance in *Drosophila* embryos: the role of hsp70 and the basis for a specific phenocopy. Ph.D. thesis. Univ. Chicago. 230 pp.
225. Krebs PA, Feder ME. 1998. Experimental manipulation of the cost of thermal acclimation in *Drosophila melanogaster*. *Biol. J. Linn. Soc.* 63:593-601
226. Hoffmann AA. 1995. Acclimation: increasing survival at a cost. *Trends Ecol. Evol.* 10:1-2
227. Calow P. 1991. Physiological costs of combating chemical toxicants: ecological implications. *Comp. Biochem. Physiol.* 100C:3-6
228. Koehn RK, Bayne BL. 1989. Towards a physiological and genetical understanding of the energetics of the stress response. *Biol. J. Linn. Soc.* 37:157-71
229. Dorner AJ, Krane MG, Kaufman RJ. 1988. Reduction of endogenous GRP78 levels improves secretion of a heterologous protein in CHO cells. *Mol. Cell Biol.* 8:4063-70
230. Dorner AJ, Wasley LC, Kaufman RJ. 1992. Overexpression of GRP78 mitigates stress induction of glucose regulated proteins and blocks secretion of selective proteins in Chinese hamster ovary cells. *EMBO J.* 11:1563-71
231. Dorner AJ, Kaufman RJ. 1994. The levels of endoplasmic reticulum proteins and ATP affect folding and secretion of selective proteins. *Biologicals* 22:103-12
232. Ryan C, Stevens TH, Schlesinger MJ. 1992. Inhibitory effects of HSP70 chaperones on nascent polypeptides. *Protein Sci.* 1:980-85
233. Heckathorn SA, Poeller GJ, Coleman JS, Hallberg RL. 1996. Nitrogen availability alters patterns of accumulation of heat stress-induced proteins in plants. *Oecologia* 105:413-18
234. Heckathorn SA, Poeller GJ, Coleman JS, Hallberg RL. 1996. Nitrogen availability and vegetative development influence the response of ribulose 1,5-bisphosphate carboxylase/oxygenase, phosphoenolpyruvate carboxylase, and heat-shock protein content to heat stress in *Zea mays* L. *Int. J. Plant Sci.* 157:588-95
235. Dietz TJ, Somero GN. 1993. Species- and tissue-specific synthesis patterns for heat-shock proteins hsp70 and hsp90 in several marine teleost fishes. *Physiol. Zool.* 66:863-80
236. Abravaya K, Phillips B, Morimoto RI. 1991. Attenuation of the heat shock response in HeLa cells is mediated by the release of bound heat shock transcription factor and is modulated by changes in growth and in heat shock temperatures. *Genes Dev.* 5:2117-27
237. Sarge KD, Cullen KE. 1997. Regulation of hsp expression during rodent spermatogenesis. *Cell. Mol. Life Sci.* 53:191-97
238. Koban M, Graham G, Prosser CL. 1987. Induction of heat-shock protein synthesis in teleost hepatocytes: effects of acclimation temperature. *Physiol. Zool.* 60:290-96
239. Deleted in proof
240. Favatier F, Bornman L, Hightower LE, Gnther E, Polla BS. 1997. Variation in hsp gene expression and Hsp polymorphism: Do they contribute

to differential disease susceptibility and stress tolerance? *Cell Stress Chaperones* 2:141-55

241. Engman DM, Sias SR, Gabe JD, Donelson JE, Dragon EA. 1989. Comparison of HSP70 genes from two strains of *Trypanosoma cruzi*. *Mol. Biochem. Parasitol.* 37:285-87

242. Engman DM, Reddy LV, Donelson JE, Kirchhoff LV. 1987. *Trypanosoma cruzi* exhibits inter- and intra-strain heterogeneity in molecular karyotype and chromosomal gene location. *Mol. Biochem. Parasitol.* 22:115-23

243. Snutch TP, Baillie DL. 1984. A high degree of DNA strain polymorphism associated with the major heat shock gene in *Caenorhabditis elegans*. *Mol. Gen. Genet.* 195:329-35

244. Hamet P, Kaiser MA, Sun Y, Page V, Vincent M, et al. 1996. HSP27 locus cosegregates with left ventricular mass independently of blood pressure. *Hypertension* 28:1112-17

245. Wallich R, Helmes C, Schaible UE, Lobet Y, Moter SE, et al. 1992. Evaluation of genetic divergence among *Borrelia burgdorferi* isolates by use of OspA, fla, HSP60, and HSP70 gene probes. *Infect. Immun.* 60:4856-66

246. Hamet P, Kong D, Pravenec M, Kunes J, Kren V, et al. 1992. Restriction fragment length polymorphism of hsp70 gene, localized in the RT1 complex, is associated with hypertension in spontaneously hypertensive rats. *Hypertension* 19:611-14

247. Grosz MD, Skow LC, Stone RT. 1994. An AluI polymorphism at the bovine 70 kD heat-shock protein-1 (HSP70-1) locus. *Anim. Genet.* 25:196

248. Jorgensen JA, Nguyen HT. 1995. Genetic analysis of heat shock proteins in maize. *Theor. Appl. Genet.* 91:38-46

249. Ottaviano E, Sari Gorla M, Pe E, Frova C. 1991. Molecular markers, RFLPs and Hsps for the genetic dissection of thermotolerance in maize. *Theor. Appl. Genet.* 81:713-19

250. Dimascio JA, Sweeney PM, Danneberger TK, Kamalay JC. 1994. Analysis of heat shock response in perennial ryegrass using maize heat shock protein clones. *Crop Sci.* 34:798-804

251. Goldschmidt-Clermont M. 1980. Two genes for the major heat-shock protein of *Drosophila melanogaster* arranged as an inverted repeat. *Nucleic Acids Res.* 8:235-52

252. Ish-Horowicz D, Pinchin SM. 1980. Genomic organization of the 87A7 and 87C1 heat-induced loci of *Drosophila melanogaster*. *J. Mol. Biol.* 142:231-45

253. Leigh-Brown AJ, Ish-Horowicz D. 1981. Evolution of the 87A and 87C heat-shock loci in *Drosophila*. *Nature* 290:677-82

254. McKechnie SW, Halford MM, McColl G, Hoffmann AA. 1998. Both allelic variation and expression of nuclear and cytoplasmic transcripts of hsr-omega are closely associated with thermal phenotype in *Drosophila*. *Proc. Natl. Acad. Sci. USA* 95:2423-28

255. Ish-Horowicz D, Pinchin SM, Schedl P, Artavanis-Tsakonas S, Mirault ME. 1979. Genetic and molecular analysis of the 87A7 and 87C1 heat-inducible loci of *D. melanogaster*. *Cell* 18:1351-58

256. Lis JT, Ish-Horowicz D, Pinchin SM. 1981. Genomic organization and transcription of the alpha beta heat shock DNA in *Drosophila melanogaster*. *Nucleic Acids Res.* 9:5297-310

257. Lis JT, Prestidge L, Hogness DS. 1978. A novel arrangement of tandemly repeated genes at a major heat shock site in *D. melanogaster*. *Cell* 14:901-19

258. Mirault ME, Goldschmidt-Clermont M, Artavanis-Tsakonas S, Schedl P. 1979. Organization of the multiple genes for the 70,000-dalton heat-shock protein in *Drosophila melanogaster*. *Proc. Natl. Acad. Sci. USA* 76:5254-58

259. Craig EA, McCarthy BJ, Wadsworth SC. 1979. Sequence organization of two recombinant plasmids containing genes for the major heat shock induced protein of *D. melanogaster*. *Cell* 16:575-88

260. Artavanis-Tsakonas S, Schedl P, Mirault ME, Moran L, Lis J. 1979. Genes for the 70,000 dalton heat shock protein in two cloned *D. melanogaster* DNA segments. *Cell* 17:9-18

261. Holmgren R, Livak K, Morimoto R, Freund R, Meselson M. 1979. Studies of cloned sequences from four *Drosophila* heat shock loci. *Cell* 18:1359-70



262. Sun CW, Griffen S, Callis J. 1997. A model for the evolution of polyubiquitin genes from the study of *Arabidopsis thaliana* ecotypes. *Plant Mol. Biol.* 34:745-58
263. Ristic Z, Gifford DJ, Cass DD. 1991. Heat shock proteins in two lines of *Zea mays* L. that differ in drought and heat resistance. *Plant Physiol.* 97:1430-34
264. Colombo SJ, Colclough ML, Timmer VR, Blumwald E. 1992. Clonal variation in heat tolerance and heat shock protein expression in black spruce. *Silvae Genet.* 41:234-39
265. Fender SE, O'Connell MA. 1989. Heat shock protein expression in thermotolerant and thermosensitive lines of cotton. *Plant Cell Rep.* 8:37-40
266. Malayer JR, Hansen PJ. 1990. Differences between Brahman and Holstein cows in heat-shock induced alterations of protein synthesis and secretion by oviducts and uterine endometrium. *J. Anim. Sci.* 68:266-80
267. Otsuka Y, Takano TS, Yamazaki T. 1997. Genetic variation in the expression of the six hsp genes in the presence of heat shock in *Drosophila melanogaster*. *Genes Genetic Syst.* 72:19-24
268. Jorgensen JA, Weng J, Ho THD, Nguyen HT. 1992. Genotype-specific heat shock proteins in two maize inbreds. *Plant Cell Rep.* 11:576-80
269. Frova C, Gorla MS. 1993. Quantitative expression of maize Hsps: genetic dissection and association with thermotolerance. *Theor. Appl. Genet.* 86:213-20
270. Weng J, Nguyen HT. 1992. Differences in the heat-shock response between thermotolerant and thermosusceptible cultivars of hexaploid wheat. *Theor. Appl. Genet.* 84:941-46
271. Brown DC, Bradley BP, Tedengren M. 1995. Genetic and environmental regulation of HSP70 expression. *Mar. Env. Res.* 39:181-84
272. Caruso M, Sacco M, Medoff G, Maresca B. 1987. Heat shock 70 gene is differentially expressed in *Histoplasma capsulatum* strains with different levels of thermotolerance and pathogenicity. *Mol. Microbiol.* 1:151-58
273. Lyashko VN, Vikulova VK, Chernikov VG, Ivanov VI, Ulmasov KA, et al. 1994. Comparison of the heat shock response in ethnically and ecologically different human populations. *Proc. Natl. Acad. Sci. USA* 91:12492-95
274. Bettencourt BR, Feder ME, Cavicchi S. 1997. Laboratory evolution of Hsp70 expression in *Drosophila melanogaster*: functional consequences and molecular bases. *Am. Zool.* 37:189A (Abstr.)
275. McColl G, Hoffmann AA, McKechnie SW. 1996. Response of two heat shock genes to selection for knockdown heat resistance in *Drosophila melanogaster*. *Genetics* 143:1615-27
276. Fender SE, O'Connell MA. 1990. Expression of the heat shock response in a tomato interspecific hybrid is not intermediate between the two parental responses. *Plant Physiol.* 93:1140-46
277. Gaugler R, Wilson M, Shearer P. 1997. Field release and environmental fate of a transgenic entomopathogenic nematode. *Biol. Control* 9:75-80
278. Hashmi S, Hashmi G, Glazer I, Gaugler R. 1998. Thermal response of *Heterorhabditis bacteriophora* transformed with the *Caenorhabditis elegans* hsp70 encoding gene. *J. Exp. Zool.* 281:164-70
279. Trent JD. 1996. A review of acquired thermotolerance, heat-shock proteins, and molecular chaperones in Archaea. *FEMS Micro. Rev.* 18:249-58
280. Holden JF, Baross JA. 1993. Enhanced thermotolerance and temperature-induced changes in protein composition in the hyperthermophilic archaeon ES4. *J. Bacteriol.* 175:2839-43
281. Hamilton PT, Reeve JN. 1985. Structure of genes and an insertion element in the methane producing archaeobacterium *Methanobrevibacter smithii*. *Mol. Gen. Genet.* 200:47-59
282. Waldmann T, Nimmesgern E, Nitsch M, Peters J, Pfeifer G, et al. 1995. The thermosome of *Thermoplasma acidophilum* and its relationship to the eukaryotic chaperonin TRiC. *Eur. J. Biochem.* 227:848-56
283. Phipps BM, Typke D, Heger R, Volker S, Hoffmann A, et al. 1993. Structure of a molecular chaperone from a thermophilic archaeobacterium. *Nature* 361:475-77
284. Nitsch M, Klumpp M, Lupas A, Baumeister W. 1997. The thermosome:

- alternating alpha and beta-subunits within the chaperonin of the archaeon *Thermoplasma acidophilum*. *J. Mol. Biol.* 267:142-49
285. Lewis VA, Hynes GM, Zheng D, Saibil H, Willison K. 1992. T-complex polypeptide-1 is a subunit of a heteromeric particle in the eukaryotic cytosol. *Nature* 358:249-52
286. Trent JD, Nimmesgern E, Wall JS, Hartl FU, Horwich AL. 1991. A molecular chaperone from a thermophilic archaeobacterium is related to the eukaryotic protein t-complex polypeptide-1. *Nature* 354:490-93
287. Hendrick JP, Hartl FU. 1995. The role of molecular chaperones in protein folding. *FASEB J.* 9:1559-69
288. Eggers DK, Welch WJ, Hansen WJ. 1997. Complexes between nascent polypeptides and their molecular chaperones in the cytosol of mammalian cells. *Mol. Biol. Cell* 8:1559-73
289. Burston SG, Clarke AR. 1995. Molecular chaperones: physical and mechanistic properties. *Essays Biochem.* 29:125-36
290. Trent JD, Kagawa HK, Yaoi T, Olle E, Zaluzec NJ. 1997. Chaperonin filaments: the archaeal cytoskeleton? *Proc. Natl. Acad. Sci. USA* 94:5383-88
291. Dascher CC, Poddar SK, Maniloff J. 1990. Heat shock response in mycoplasmas, genome-limited organisms. *J. Bacteriol.* 172:1823-27
292. Gupta RS, Singh B. 1994. Phylogenetic analysis of 70 kD heat shock protein sequences suggests a chimeric origin for the eukaryotic cell nucleus. *Curr. Biol.* 4:1104-14
293. Bustard K, Gupta RS. 1997. The sequences of heat shock protein 40 (DanJ) homologs provide evidence for a close evolutionary relationship between the *Deinococcus thermus* group and Cyanobacteria. *J. Mol. Evol.* 45:193-205
294. Gupta RS, Bustard K, Falah M, Singh D. 1997. Sequencing of heat shock protein 70 (DnaK) homologs from *Deinococcus proteolyticus* and *Thermomicrobium roseum* and their integration in a protein-based phylogeny of prokaryotes. *J. Bacteriol.* 179:345-57
295. Gupta RS. 1995. Phylogenetic analysis of the 90 kD heat shock family of protein sequences and an examination of the relationship among animals, plants, and fungi species. *Mol. Biol. Evol.* 12:1063-73
296. Gupta RS, Aitken K, Falah M, Singh B. 1994. Cloning of *Giardia lamblia* heat shock protein HSP70 homologs: implications regarding origin of eukaryotic cells and of endoplasmic reticulum. *Proc. Natl. Acad. Sci. USA* 91:2895-99
297. Gupta RS, Singh B. 1992. Cloning of the HSP70 gene from *Halobacterium marismortui*: relatedness of archaeobacterial HSP70 to its eubacterial homologs and a model for the evolution of the HSP70 gene. *J. Bacteriol.* 174:4594-605
298. Boorstein WR, Ziegelhoffer T, Craig EA. 1994. Molecular evolution of the HSP70 multigene family. *J. Mol. Evol.* 38:1-17
299. Rubin DM, Mehta AD, Zhu J, Shoham S, Chen X, et al. 1993. Genomic structure and sequence analysis of *Drosophila melanogaster* HSC70 genes. *Gene* 128:155-63
300. Tavaría M, Gabriele T, Kola I, Anderson RL. 1996. A hitchhiker's guide to the human Hsp70 family. *Cell Stress Chaperones* 1:23-28
301. de Jong WW, Leunissen JA, Voorter CE. 1993. Evolution of the alphacrystallin/small heat-shock protein family. *Mol. Biol. Evol.* 10:103-26
302. Ohta T. 1994. Further examples of evolution by gene duplication revealed through DNA sequence comparisons. *Genetics* 138:1331-37
303. Benedict MQ, Cockburn AF, Seawright JA. 1993. The Hsp70 heat-shock gene family of the mosquito *Anopheles albimanus*. *Insect Mol. Biol.* 2:93-102
304. Drosopoulou E, Konstantopoulou I, Scouras ZG. 1996. The heat shock genes in the *Drosophila montium* subgroup: chromosomal localization and evolutionary implications. *Chromosoma* 105:104-10
305. Pardali E, Feggou E, Drosopoulou E, Konstantopoulou I, Scouras ZG, Mavragani-Tsipidou P. 1996. The Afrotropical *Drosophila montium* subgroup: Balbiani ring 1, polytene chromosomes, and heat shock response of *Drosophila vulcana*. *Genome* 39:588-97
306. Sanchez Y, Lindquist SL. 1990. HSP104 required for induced thermotolerance. *Science* 248:1112-15
307. Feder ME, Block BA. 1991. On the future of physiological ecology.

308. Lau S, Patnaik N, Sayen MR, Mestril R. 1997. Simultaneous overexpression of two stress proteins in rat cardiomyocytes and myogenic cells confers protection against ischemia-induced injury. *Circulation* 96:2287-94
309. Huot J, Roy G, Lambert H, Chretien P, Landry J. 1991. Increased survival after treatments **with** anticancer agents of Chinese hamster cells expressing the human Mr 27,000 heat shock protein. *Cancer Res.* 51:5245-52
310. Mehlen P, Preville X, Chareyron P, Briolay J, Klemenz R, Arrigo AP. 1995. Constitutive expression of human hsp27, *Drosophila* hsp27, or human alpha B-crystallin confers resistance to TNF- and oxidative stress-induced cytotoxicity in stably transfected murine L929 fibroblasts. *J. Immunol.* 154:363-74
311. Jaattela M, Wissing D. 1993. Heat-shock proteins protect cells from monocyte cytotoxicity: possible mechanism of self-protection. *J. Exp. Med.* 177:231-36
312. Trautinger F, Kokesch C, Herbacek I, Knobler RM, Kindas-Mugge I. 1997. Overexpression of the small heat shock protein, hsp27, confers resistance to hyperthermia, but not to oxidative stress and UV-induced cell death, in a stably transfected squamous cell carcinoma cell line. *J. Photochem. Photobiol.* 39B:90-95
313. Rollet E, Lavoie JN, Landry J, Tanguay RM. 1992. Expression of *Drosophila*'s 27 kDa heat shock protein into rodent cells confers thermal resistance. *Biochem. Biophys. Res. Commun.* 185:116-20
314. Lavoie JN, Gingras-Breton G, Tanguay RM, Landry J. 1993. Induction of Chinese hamster HSP27 gene expression in mouse cells confers resistance to heat shock. HSP27 stabilization of the microfilament organization. *J. Biol. Chem.* 268:3420-29
315. Landry J, Chretien P, Lambert H, Hickey E, Weber LA. 1989. Heat shock resistance conferred by expression of the human HSP27 gene in rodent cells. *J. Cell Biol.* 109:7-15
316. Wissing D, Jaattela M. 1996. HSP27 and HSP70 increase the survival of WEHI-S cells exposed to hyperthermia. *Int. J. Hypertherm.* 12:125-38
317. Wang G, Klostergaard J, Khodadadian M, Wu J, Wu TW, et al. 1996. Murine cells transfected with human Hsp27 cDNA resist TNF-induced cytotoxicity. *J. Immunother. Emphasis Tumor. Immunol.* 19:9-20
318. Martin JL, Mestril R, Hilal-Dandan R, Brunton LL, Dillmann WH. 1997. Small heat shock proteins and protection against ischemic injury in cardiac myocytes. *Circulation* 96:4343-48
319. Stege GJ, Kampinga HH, Konings AW. 1995. Heat-induced intranuclear protein aggregation and thermal radiosensitization. *Int. J. Radiat. Biol.* 67:203-9
320. Blackburn R, Galoforo S, Berns CM, Ireland M, Cho JM, et al. 1996. Thermal response in murine L929 cells lacking alpha B-crystallin expression and alpha B-crystallin expressing L929 transfectants. *Mol. Cell. Biochem.* 155:51-60
321. van den Ijssel PR, Overkamp P, Knauf U, Gaestel M, de Jong WW. 1994. Alpha A-crystallin confers cellular thermoresistance. *FEBS Lett.* 355:54-56
322. Heads RJ, Yellon DM, Latchman DS. 1995. Differential cytoprotection against heat stress or hypoxia following expression of specific stress protein genes in myogenic cells. *J. Mol. Cell. Cardiol.* 27:1669-78
323. Cumming DV, Heads RJ, Watson A, Latchman DS, Yellon DM. 1996. Differential protection of primary rat cardiocytes by transfection of specific heat stress proteins. *J. Mol. Cell. Cardiol.* 28:2343-49
324. Lukacs KV, Nakakes A, Atkins CJ, Lowrie DB, Colston MJ. 1997. In vivo gene therapy of malignant tumours with heat shock protein-65 gene. *Gene Ther.* 4:346-50
325. Lukacs KV, Lowrie DB, Stokes RW, Colston MJ. 1993. Tumor cells transfected with a bacterial heat-shock gene lose tumorigenicity and induce protection against tumors. *J. Exp. Med.* 178:343-48
326. Wischmeyer PE, Musch MW, Madonna MB, Thisted R, Chang EB. 1997.

Glutamine protects intestinal epithelial cells: role of inducible HSP70.  
Am. J. Physiol. 272:G879-84

327. Angelidis CE, Lazaridis I, Pagoulatos GN. 1991. Constitutive expression of heat-shock protein 70 in mammalian cells confers thermoresistance. Eur. J. Biochem. 199:35-39

328. Mailhos C, Howard MK, Latchman DS. 1994. Heat shock proteins hsp90 and hsp70 protect neuronal cells from thermal stress but not from programmed cell death. J. Neurochem. 63:1787-95

329. Wyatt S, Mailhos C, Latchman DS. 1996. Trigeminal ganglion neurons are protected by the heat shock proteins hsp70 and hsp90 from thermal stress but not from programmed cell death following nerve growth factor withdrawal. Brain Res. Mol. Brain Res. 39:52-56

330. Uney JB, Staley K, Tyers P, Sofroniew MV, Kew JN. 1994. Transfection with hsp70i protects rat dorsal root ganglia neurones and glia from heat stress. Gene Ther. 1:S65

331. Solomon JM, Rossi JM, Golic K, McGarry T, Lindquist S. 1991. Changes in Hsp70 alter thermotolerance and heat-shock regulation in *Drosophila*. New Biol. 3:1106-20

332. Sato K, Saito H, Matsuki N. 1996. HSP70 is essential to the neuroprotective effect of heat-shock. Brain Res. 740:117-23

333. Riabowol KT, Mizzen LA, Welch WJ. 1988. Heat shock is lethal to fibroblasts microinjected with antibodies against hsp70. Science 242:433-36

334. Mestrlil R, Giordano FJ, Conde AG, Dillmann WH. 1996. Adenovirus-mediated gene transfer of a heat shock protein 70 (hsp 70i) protects against simulated ischemia. J. Mol. Cell. Cardiol. 28:2351-58

335. Liu RY, Li X, Li L, Li GC. 1992. Expression of human hsp70 in rat fibroblasts enhances cell survival and facilitates recovery from translational and transcriptional inhibition following heat shock. Cancer Res. 52:3667-73

336. Nakata N, Kato H, Kogure K. 1993. Inhibition of ischaemic tolerance in the gerbil hippocampus by quercetin and anti-heat shock protein-70 antibody. NeuroReport 4:695-98

337. Lee YJ, Kim D, Hou ZZ, Curetty L, Borrelli MJ, Corry PM. 1993. Alteration of heat sensitivity by introduction of hsp70 or anti-hsp70 in CHO cells. J. Therm. Biol. 18:229-36

338. Johnston RN, Kucey BL. 1988. Competitive inhibition of hsp70 gene expression causes thermosensitivity. Science 242:1551-54

339. Heads RJ, Latchman DS, Yellon DM. 1994. Stable high level expression of a transfected human HSP70 gene protects a heart-derived muscle cell line against thermal stress. J. Mol. Cell. Cardiol. 26:695-99

340. Amin V, Cumming DV, Latchman DS. 1996. Over-expression of heat shock protein 70 protects neuronal cells against both thermal and ischaemic stress but with different efficiencies. Neurosci. Lett. 206:45-48

341. Khan NA, Sotelo J. 1989. Heat shock stress is deleterious to CNS cultured neurons microinjected with anti-HSP70 antibodies. Biol. Cell 65:199-202

342. Li GC, Li LG, Liu YK, Mak JY, Chen LL, Lee WM. 1991. Thermal response of rat fibroblasts stably transfected with the human 70-kDa heat shock proteinencoding gene. Proc. Natl. Acad. Sci. USA 88:1681-85

343. Williams RS, Thomas JA, Fina M, German Z, Benjamin IJ. 1993. Human heat shock protein 70 (hsp70) protects murine cells from injury during metabolic stress. J. Clin. Invest. 92:503-8

344. Mestrlil R, Chi SH, Sayen MR, O'Reilly K, Dillmann WH. 1994. Expression of inducible stress protein 70 in rat heart myogenic cells confers protection against simulated ischemia-induced injury. J. Clin. Invest. 93:759-67

345. Dillmann WH, Mestrlil R. 1995. Heat shock proteins in myocardial stress. Z. Kardiologie. 4:87-90

346. Jacobs M, Andersen JB, Kontinen V, Sarvas M. 1993. The DNA-binding activity of the human heat shock transcription factor is regulated in vivo by Hsp70. Mol. Cell Biol. 13:5427-38

347. Ding XZ, Tsokos GC, Smallridge RC, Kiang JG. 1997. Heat shock gene-expression in HSP-70 and HSF1 gene-transfected human epidermoid A-431 cells. Mol. Cell. Biochem. 167:145-52

348. Chi SH, Mestrlil R. 1996. Stable expression of a human HSP70 gene

- in a rat myogenic cell line confers protection against endotoxin. *Am. J. Physiol.* 270:C1017-21
349. Han MY, Park YM. 1997. Reduced protein denaturation in thermotolerant cells by elevated levels of HSP70. *Korean J. Pharmacol.* 32:433-44
350. Jaattela M. 1995. Over-expression of hsp70 confers tumorigenicity to mouse fibrosarcoma cells. *Int. J. Cancer* 60:689-93
351. Wei YQ, Zhao X, Kariya Y, Teshigawara K, Uchida A. 1995. Inhibition of proliferation and induction of apoptosis by abrogation of heat-shock protein (HSP) 70 expression in tumor cells. *Cancer Immunol. Immunother.* 40:73-78
352. Karlseder J, Wissing D, Holzer G, Orel L, Sliutz G, et al. 1996. Hsp70 overexpression mediates the escape of a doxorubicin-induced G2 cell cycle arrest. *Biochem. Biophys. Res. Commun.* 220:153-59
353. Henle KJ, Jethmalani SM, Li L, Li GC. 1997. Protein glycosylation in a heat-resistant rat fibroblast cell model expressing human HSP70. *Biochem. Biophys. Res. Commun.* 232:26-32
354. Simon MM, Reikerstorfer A, Schwarz A, Krone C, Luger TA, et al. 1995. Heat shock protein 70 overexpression affects the response to ultraviolet light in murine fibroblasts. Evidence for increased cell viability and suppression of cytokine release. *J. Clin. Invest.* 95:926-33
355. Liossis SN, Ding XZ, Kiang JG, Tsokos GC. 1997. Overexpression of the heat shock protein 70 enhances the TCR/CD3- and Fas/Apo-1/CD95-mediated apoptotic cell death in Jurkat T cells. *J. Immunol.* 158:5668-75
356. Mosser DD, Caron AW, Bourget L, Denis-Larose C, Massie B. 1997. Role of the human heat shock protein hsp70 in protection against stress-induced apoptosis. *Mol. Cell. Biol.* 17:5317-27
357. Suzuki K, Sawa Y, Kaneda Y, Ichikawa H, Shirakura R, Matsuda H. 1997. In vivo gene transfection with heat shock protein 70 enhances myocardial tolerance to ischemia-reperfusion injury in rat. *J. Clin. Invest.* 99:1645-50
358. Plumier JC, Ross BM, Currie RW, Angelidis CE, Kazlaris H, et al. 1995. Transgenic mice expressing the human heat shock protein 70 have improved post-ischemic myocardial recovery. *J. Clin. Invest.* 95:1854-60
359. Marber MS, Mestrlil R, Chi SH, Sayen MR, Yellon DM, Dillmann WH. 1995. Overexpression of the rat inducible 70-kD heat stress protein in a transgenic mouse increases the resistance of the heart to ischemic injury. *J. Clin. Invest.* 95:1446-56
360. Plumier JC, Krueger AM, Currie RW, Kontoyiannis D, Kollias G, Pagoulatos GN. 1997. Transgenic mice expressing the human inducible Hsp70 have hippocampal neurons resistant to ischemic injury. *Cell Stress Chaperones* 2:162-67
361. Lee JH, Schoffl F. 1996. An Hsp70 antisense gene affects the expression of HSP70/HSC70, the regulation of HSF, and the acquisition of thermotolerance in transgenic *Arabidopsis thaliana*. *Mol. Gen. Genet.* 252:11-19
362. Galea-Lauri J, Richardson AJ, Latchman DS, Katz DR. 1996. Increased heat shock protein 90 (hsp90) expression leads to increased apoptosis in the monoblastoid cell line U937 following induction with TNF-alpha and cycloheximide: a possible role in immunopathology. *J. Immunol.* 157:4109-18
363. Nakano M, Mann DL, Knowlton AA. 1997. Blocking the endogenous increase in HSP 72 increases susceptibility to hypoxia and reoxygenation in isolated adult feline cardiocytes. *Circulation* 95:1523-31
364. Hutter JJ, Mestrlil R, Tam EK, Sievers RE, Dillmann WH, Wolfe CL. 1996. Overexpression of heat shock protein 72 in transgenic mice decreases infarct size in vivo. *Circulation* 94:1408-11
365. Galea-Lauri J, Latchman DS, Katz DR. 1996. The role of the 90-kDa heat shock protein in cell cycle control and differentiation of the monoblastoid cell line U937. *Exp. Cell Res.* 226:243-54
366. Schirmer EC, Lindquist S, Vierling E. 1994. An *Arabidopsis* heat shock protein complements a thermotolerance defect in yeast. *Plant Cell* 6:1899-909
367. Kutsikova IUA, Mamon LA. 1996. Consequences of exposure to extreme conditions in somatic cells of *Drosophila melanogaster* under conditions of

disturbed synthesis of heat shock proteins. Genetika 32:1406-16

368. Mamon LA, Kutsikova YA. 1993. The role of the heat-shock proteins in recovery of high temperature induced damages of mitotic chromosomes in *Drosophila melanogaster*. Genetika 29:604-12

369. Mamon LA, Kutsikova YA. 1993. The role of the heat-shock proteins in recovery of cell proliferation following high temperature treatment of *Drosophila melanogaster*. Genetika 29:791-98

370. Koishi M, Hosokawa N, Sato M, Nakai A, Hirayoshi K, et al. 1992. Quercetin, an inhibitor of heat shock protein synthesis, inhibits the acquisition of thermotolerance in a human colon carcinoma cell line. Jpn. J. Cancer Res. 83:1216-22

371. Lee YJ, Curetty L, Hou ZZ, Kim SH, Kim JH, Corry PM. 1992. Effect of pH on quercetin-induced suppression of heat shock gene expression and thermotolerance development in HT-29 cells. Biochem. Biophys. Res. Commun. 186:1121-28

372. Jedlicka P, Mortin MA, Wu C. 1997. Multiple functions of *Drosophila* heat shock transcription factor in vivo. EMBO J. 16:2452-62

373. Lee JH, Hubel A, Schoffl F. 1995. Derepression of the activity of genetically engineered heat shock factor causes constitutive synthesis of heat shock proteins and increased thermotolerance in transgenic *Arabidopsis*. Plant J. 8:603-12

10/7/14 (Item 2 from file: 98)  
DIALOG(R) File 98:General Sci Abs/Full-Text  
(c) 2002 The HW Wilson Co. All rts. reserv.

03760145 H.W. WILSON RECORD NUMBER: BGS198010145 (THIS IS THE FULLTEXT)  
Emerging foodborne diseases: an evolving public health challenge.

Tauxe, Robert V

Emerging Infectious Diseases (Emerging Infect Dis) v. 3 (Oct./Dec. '97) p. 425-34

LANGUAGE: English

COUNTRY OF PUBLICATION: United States

WORD COUNT: 6991

ABSTRACT: The epidemiology of foodborne disease is changing. New pathogens have emerged, and some have spread worldwide. Many, including *Salmonella*, *Escherichia coli* O157:H7, *Campylobacter*, and *Yersinia enterocolitica*, have reservoirs in healthy food animals, from which they spread to an increasing variety of foods. These pathogens cause millions of cases of sporadic illness and chronic complications, as well as large and challenging outbreaks over many states and nations. Improved surveillance that combines rapid subtyping methods, cluster identification, and collaborative epidemiologic investigation can identify and halt large, dispersed outbreaks. Outbreak investigations and case-control studies of sporadic cases can identify sources of infection and guide the development of specific prevention strategies. Better understanding of how pathogens persist in animal reservoirs is also critical to successful long-term prevention. In the past, the central challenge of foodborne disease lay in preventing the contamination of human food with sewage or animal manure. In the future, prevention of foodborne disease will increasingly depend on controlling contamination of feed and water consumed by the animals themselves. Reprinted by permission of the publisher.

TEXT:

Every year, in the United States foodborne infections cause millions of illnesses and thousands of deaths; most infections go undiagnosed and unreported. As the epidemiology of foodborne infections evolves, old scenarios and solutions need to be updated. This article reviews main trends in the evolution of foodborne disease epidemiology and their effect on surveillance and prevention activities.

Preventing foodborne disease is a multifaceted process, without simple and universal solutions. For most foodborne pathogens, no **vaccines** are available. Consumer education about basic principles of food safety, an

important component of prevention, by itself is insufficient. Food reaches the consumer through long chains of industrial production, in which many opportunities for contamination exist. The general strategy of prevention is to understand the mechanisms by which contamination and disease transmission can occur well enough to interrupt them. An outbreak investigation or epidemiologic study should go beyond identifying a suspected food and pulling it from the shelf to defining the chain of events that allowed contamination with an organism in large enough numbers to cause illness. We learn from the investigation what went wrong, in order to devise strategies to prevent similar events in the future. Although outbreaks make the news, most foodborne infections occur as individual or sporadic cases. Therefore, the sources of sporadic cases must also be investigated and understood.

#### EMERGING FOODBORNE PATHOGENS

Substantial progress has been made in preventing foodborne diseases. For example, typhoid fever, extremely common at the beginning of the 20th century, is now almost forgotten in the United States. It was conquered in the preantibiotic era by disinfection of drinking water, sewage **treatment**, milk sanitation and pasteurization, and shellfish bed sanitation (Figure 1). Similarly, cholera, bovine tuberculosis, and trichinosis have also been controlled in the United States. However, new foodborne pathogens have emerged. Among the first of these were infections caused by nontyphoid strains of *Salmonella*, which have increased decade by decade since World War II (Figure 1). In the last 20 years, other infectious agents have been either newly described or newly associated with foodborne transmission (Table 1). *Vibrio vulnificus*, *Escherichia coli* O157:H7, and *Cyclospora cayetanensis* are examples of newly described pathogens that often are foodborne. *V. vulnificus* was identified in the bloodstream of persons with underlying liver disease who had fulminant infections after eating raw oysters or being exposed to seawater; this organism lives in the sea and can be a natural summertime commensal organism in shellfish (1). *E. coli* O157:H7 was first identified as a pathogen in 1982 in an outbreak of bloody diarrhea traced to hamburgers from a fast-food chain (2); it was subsequently shown to have a reservoir in healthy cattle (3). *Cyclospora*, known previously as a cyanobacterial-like organism, received its current taxonomic designation in 1992 and emerged as a foodborne pathogen in outbreaks traced to imported Guatemalan raspberries in 1996 (4,5). The similarity of *Cyclospora* to *Eimeria* coccidian pathogens of birds suggests an avian reservoir (4,5).

Some known pathogens have only recently been shown to be predominantly foodborne. For example, *Listeria monocytogenes* was long known as a cause of meningitis and other invasive infections in immunocompromised hosts. How these hosts became infected remained unknown until a series of investigations identified food as the most common source (6). Similarly, *Campylobacter jejuni* was known as a rare opportunistic bloodstream infection until veterinary diagnostic methods used on specimens from humans showed it was a common cause of diarrheal illness (7). Subsequent epidemiologic investigations implicated poultry and raw milk as the most common sources of sporadic cases and outbreaks, respectively (8). *Yersinia enterocolitica*, rare in the United States but a common cause of diarrheal illness and pseudoappendicitis in northern Europe and elsewhere, is now known to be most frequently associated with undercooked pork (9).

These foodborne pathogens share a number of characteristics. Virtually all have an animal reservoir from which they spread to humans; that is, they are foodborne zoonoses. In marked contrast to many established zoonoses, these new zoonoses do not often cause illness in the infected host animal. The chicken with lifelong ovarian infection with *Salmonella* serotype Enteritidis, the calf carrying *E. coli* O157:H7, and the oyster carrying Norwalk virus or *V. vulnificus* appear healthy; therefore, public health concerns must now include apparently healthy animals. Limited existing research on how animals acquire and transmit emerging pathogens among themselves often implicates contaminated fodder and water; therefore, public health concerns must now include the safety of what food animals

themselves eat and drink.

For reasons that remain unclear, these pathogens can rapidly spread globally. For example, *Y. enterocolitica* spread globally among pigs in the 1970s (10); *Salmonella* serotype Enteritidis appeared simultaneously around the world in the 1980s (11); and *Salmonella* Typhimurium Definitive Type (DT) 104 is now appearing in North America, Europe, and perhaps elsewhere (12); therefore, public health concerns must now include events happening around the world, as harbingers of what may appear here.

Many emerging zoonotic pathogens are becoming increasingly resistant to antimicrobial agents, largely because of the widespread use of antibiotics in the animal reservoir. For example, *Campylobacter* isolated from human patients in Europe is now increasingly resistant to fluoroquinolones, after these agents were introduced for use in animals (13). *Salmonellae* have become increasingly resistant to a variety of antimicrobial agents in the United States (14); therefore, public health concerns must include the patterns of antimicrobial use in agriculture as well as in human medicine.

The foods contaminated with emerging pathogens usually look, smell, and taste normal, and the pathogen often survives traditional preparation techniques: *E. coli* O157:H7 in meat can survive the gentle heating that a rare hamburger gets (15); *Salmonella* Enteritidis in **eggs** survives in an omelette (16); and Norwalk virus in oysters survives gentle steaming (17). Following standard and traditional recipes can cause illness and outbreaks. Contamination with the new foodborne zoonoses eludes traditional food inspection, which relies on visual identification of foodborne hazards. These pathogens demand new control strategies, which would minimize the likelihood of contamination in the first place. The rate at which new pathogens have been identified suggests that many more remain to be discovered. Many of the foodborne infections of the future are likely to arise from the animal reservoirs from which we draw our food supply.

Once a new foodborne disease is identified, a number of critical questions need to be answered to develop a rational approach to prevention: What is the nature of the disease? What is the nature of the pathogen? What are simple ways to easily identify the pathogen and diagnose the disease? What is the incidence of the infection? How can the disease be **treated**? Which foods transmit the infection? How does the pathogen get into the food, and how well does it persist there? Is there an animal reservoir? How do the animals themselves become infected? How can the disease be prevented? Does the prevention strategy work?

The answers to these questions do not come rapidly. Knowledge accumulates gradually, as a result of detailed scientific investigations, often conducted during outbreaks (18). After 15 years of research, we know a great deal about infections with *E. coli* O157:H7, but we still do not know how best to **treat** the infection, nor how the cattle (the principal source of infection for humans) themselves become infected. Better slaughter procedures and pasteurization of milk are useful control strategies for this pathogen in meat and milk, as irradiation of meat may be in the future. More needs to be learned: for example, it remains unclear how best to prevent this organism from contaminating lettuce or apple juice. For more recently identified agents, even less is known.

#### NEW FOOD VEHICLES OF TRANSMISSION

Along with new pathogens, an array of new food vehicles of transmission have been implicated in recent years. Traditionally, the food implicated in a foodborne outbreak was undercooked meat, poultry or seafood, or unpasteurized milk. Now, additional foods previously thought safe are considered hazardous. For example, for centuries, the internal contents of an **egg** were presumed safe to eat raw. However, epidemic *Salmonella* Enteritidis infection among **egg**-laying flocks indicates that intact **eggs** may have internal contamination with this *Salmonella* serotype. Many outbreaks are caused by contaminated shell **eggs**, including **eggs** used in such traditional recipes as **eggnog** and Caesar salad, lightly cooked **eggs** in omelettes and French toast, and even foods one would presume thoroughly cooked, such as lasagna and meringue pie (19,20). *E. coli* O157:H7 has caused illness through an ever-broadening



spectrum of foods, beyond the beef and raw milk that are directly related to the bovine reservoir. In 1992, an outbreak caused by apple cider showed that this organism could be transmitted through a food with a pH level of less than 4.0, possibly after contact of fresh produce with manure (21). A recent outbreak traced to venison jerky suggests a wild deer reservoir, so both cattle and feral deer manure are of concern (22). Imported raspberries contaminated with *Cyclospora* caused an epidemic in the United States in 1996, possibly because contaminated surface water was used to spray the berries with fungicide before harvest (5). Norwalk-like viruses, which appear to have a human reservoir, have contaminated oysters harvested from pristine waters by oyster catchers who did not use toilets with holding tanks on their boats and were themselves the likely source of the virus (23).

The new food vehicles of disease share several features. Contamination typically occurs early in the production process, rather than just before consumption. Because of consumer demand and the global food market, ingredients from many countries may be combined in a single dish, which makes the specific source of contamination difficult to trace. These foods have fewer barriers to microbial growth, such as salt, sugar, or preservatives; therefore, simple transgressions can make the food unsafe. Because the food has a short shelf life, it may often be gone by the time the outbreak is recognized; therefore, efforts to prevent contamination at the source are very important.

An increasing, though still limited, proportion of reported foodborne outbreaks are being traced to fresh produce (24). A series of outbreaks recently investigated by the Centers for Disease Control and Prevention (CDC) has linked a variety of pathogens to fresh fruits and vegetables harvested in the United States and elsewhere (Table 2). The investigations have often been triggered by detection of more cases than expected of a rare serotype of *Salmonella* or *Shigella* or by diagnosis of a rare infection like cyclosporiasis. Outbreaks caused by common serotypes are more likely to be missed. Various possible points of contamination have been identified during these investigations, including contamination during production and harvest, initial processing and packing, distribution, and final processing (Table 3). For example, fresh or inadequately composted manure is used sometimes, although *E. coli* O157:H7 has been shown to survive for up to 70 days in bovine feces (25). Untreated or contaminated water seems to be a particularly likely source of contamination. Water used for spraying, washing, and maintaining the appearance of produce must be microbiologically safe. After two large outbreaks of salmonellosis were traced to imported cantaloupe, the melon industry considered a "Melon Safety Plan," focusing particularly on the chlorination of water used to wash melons and to make ice for shipping them. Although the extent to which the plan was implemented is unknown, no further large outbreaks have occurred. After two large outbreaks of salmonellosis were traced to a single tomato packer in the Southeast, an automated chlorination system was developed for the packing plant wash tank. Because tomatoes absorb water (and associated bacteria) if washed in water colder than they are, particular attention was also focused on the temperature of the water bath (26,27). No further outbreaks have been linked to southeastern tomatoes. Similar attention is warranted for water used to rinse lettuce heads in packing sheds and to crisp them in grocery stores as well as for water used in processing other fresh produce.

#### A NEW OUTBREAK SCENARIO

Because of changes in the way food is produced and distributed, a new kind of outbreak has appeared. The traditional foodborne outbreak scenario often follows a church supper, family picnic, wedding reception, or other social event. This scenario involves an acute and highly local outbreak, with a high inoculum dose and a high attack rate. The outbreak is typically immediately apparent to those in the local group, who promptly involve medical and public health authorities. The investigation identifies a food-handling error in a small kitchen that occurs shortly before consumption. The solution is also local. Such outbreaks still occur, and

handling them remains an important function of a local health department.

However, diffuse and widespread outbreaks, involving many counties, states, and even nations (28), are identified more frequently and follow an entirely different scenario. The new scenario is the result of low-level contamination of a widely distributed commercial food product. In most jurisdictions, the increase in cases may be inapparent against the background illness. The outbreak is detected only because of a fortuitous concentration of cases in one location, because the pathogen causing the outbreak is unusual, or because laboratory-based subtyping of strains collected over a wide area identifies a diffuse surge in one subtype. In such outbreaks, investigation can require coordinated efforts of a large team to clarify the extent of the outbreak, implicate a specific food, and determine the source of contamination. Often, no obvious terminal food-handling error is found. Instead, contamination is the result of an event in the industrial chain of food production. Investigating, controlling, and preventing such outbreaks can have industrywide implications.

These diffuse outbreaks can be caused by a variety of foods. Because fresh produce is usually widely distributed, most of the produce-related outbreaks listed in Table 2 were multistate events. Some of the largest outbreaks affected most states at once. For example, a recent outbreak of *Salmonella* Enteritidis infections caused by a nationally distributed brand of ice cream affected the entire nation (29). Although it caused an estimated 250,000 illnesses, it was detected only when vigorous routine surveillance identified a surge in reported infections with *S. Enteritidis* in one area of southern Minnesota. The consumers affected did not make food-handling errors with their ice cream, so food safety instruction could not have prevented this outbreak. The ice cream premix was transported after pasteurization to the ice cream factory in tanker trucks that had been used to haul raw **eggs**. The huge epidemic was the result of a basic failure on an industrial scale to separate the raw from the cooked.

*S. Enteritidis* infections also illustrate why surveillance and investigation of sporadic cases are needed. A diffuse increase in sporadic cases can occur well before a local or large outbreak focuses attention on the emergence of a pathogen. The isolation rate for *S. Enteritidis* began to increase sharply in the New England region in 1978 (Figure 2); all cases were sporadic. In 1982, an outbreak in a New England nursing home was traced to **eggs** from a local supplier. However, the **egg** connection was not really appreciated until 1986, when a large multistate outbreak of *S. Enteritidis* infections was traced to stuffed pasta made with raw **eggs** and labeled "fully cooked." This outbreak, affecting an estimated 3,000 persons in seven states, led to the documentation that *S. Enteritidis* was present on **egg**-laying farms and to the subsequent demonstration that both outbreaks and sporadic cases of infections were associated with shell **eggs** (19,30). Since then, *Enteritidis* has become the most common serotype of *Salmonella* isolated in the United States, accounting for 25[percent] of all *Salmonella* reported in the country and causing outbreaks coast to coast. **Eggs** remain the dominant source of these infections, causing large outbreaks when they are pooled and undercooked and individual sporadic cases among consumers who eat individual **eggs** (20,31). Perhaps focused investigation and control measures taken when the localized increase in sporadic *Salmonella* cases was just beginning might have prevented the subsequent spread.

#### CHANGING SURVEILLANCE STRATEGIES

In the United States, surveillance for diseases of major public health importance has been conducted for many years. The legal framework for surveillance resides in the state public health epidemiology offices, which share data with CDC. The first surveillance systems depended on physician or coroner notification of specific diseases and conditions, with reports going first to the local health department, then to state and federal offices. Now electronic, this form of surveillance is still used for many specific conditions (32). In 1962, a second channel was developed specifically for *Salmonella*, to take advantage of the added public health

information provided by subtyping the strains of bacteria (33). Clinical laboratories that isolated *Salmonella* from humans were requested or required to send the strains to the state public health laboratory for serotyping. Although knowing the serotype is usually of little benefit to the individual patient, it has been critical to protecting and improving the health of the public at large. Serotyping allows cases that might otherwise appear unrelated to be included in an investigation because they are of the same serotype. Moreover, infections that are close in time and space to an outbreak but are caused by nonoutbreak serotypes and are probably unrelated can be discounted. Results of serotyping are now sent electronically from public health laboratories and can be rapidly analyzed and summarized. *Salmonella* serotyping was the first subtype-based surveillance system and is a model for similar systems (34). Yet another source of surveillance data involves summary reports of foodborne disease outbreak investigations from local and state health departments (35). About 400 such outbreaks are reported annually, by a system that remains paper-based, labor-intensive, and slow.

Existing surveillance systems provide a limited and relatively inexpensive net for tracing large-scale trends in foodborne diseases under surveillance and for detecting outbreaks of established pathogens in the United States. However, they are less sensitive to diffuse outbreaks of common pathogens, provide little detail on sporadic cases, and are not easy to extend to emerging pathogens. In the future, changes in health delivery may impinge on the way that diagnoses are made and reported, leading to artifactual changes in reported disease incidence.

Therefore, CDC, in collaboration with state health departments and federal food regulatory agencies, is enhancing national surveillance for foodborne diseases in several ways. First, the role of subtyping in public health laboratories is being expanded to encompass new molecular subtyping methods. Beginning in 1997, a national subtyping network for *E. coli* O157:H7 of participating state public health department laboratories and CDC will use a single standardized laboratory protocol to subtype strains of this important pathogen. The standard method, pulsed-field gel electrophoresis, can be easily adapted to other bacterial pathogens. In this network, each participating laboratory will be able to routinely compare the genetic gel patterns of strains of *E. coli* O157:H7 with the patterns in a national pattern bank. This will enable rapid detection of clusters of related cases within the state and will focus investigative resources on the cases most likely to be linked. It will also enable related cases scattered across several states to be linked so that a common source can be sought.

Another surveillance strategy, now implemented, is active surveillance in sentinel populations. Since January 1996, at five U.S. sentinel sites, additional surveillance resources make it possible to contact laboratories directly for regular reporting of bacterial infections likely to be foodborne (36; Figure 3). In addition, surveys of the population, physicians, and laboratories measure the proportion of diarrheal diseases that are undiagnosed and unreported so that the true disease incidence can be estimated. This surveillance, known as FoodNet, is the platform on which more detailed investigations, including case-control studies of sporadic cases of common foodborne infections, are being conducted.

Yet another new surveillance initiative is the routine monitoring of antimicrobial resistance among a sample of *Salmonella* and *E. coli* O157:H7 bacteria isolated from humans (37). A new cluster detection algorithm is being applied routinely to surveillance data for *Salmonella* at the national level, making it possible to detect and flag possible outbreaks as soon as the data are reported (38). Implementation of such algorithms for other infections and at the state level will further increase the usefulness of routine surveillance.

Further enhancements are possible as active surveillance through FoodNet is extended to a wider spectrum of infections, including foodborne parasitic and viral infections. In 1997, active surveillance for *Cyclospora* began in FoodNet, which quickly resulted in the detection of a diffuse outbreak among persons who had been on a Caribbean cruise ship that made

stops in Mexico and Central America (CDC, unpub. data). Application of standardized molecular subtyping methods to other foodborne pathogens will provide a more sensitive warning system for diffuse outbreaks of a variety of pathogens. To handle outbreaks in areas not covered by FoodNet, standard surveillance and investigative capacities in state health department epidemiology offices and laboratories should be strengthened. In addition, enhanced international consultation will be critical to better detect and investigate international or global outbreaks (28).

#### IMPLICATIONS OF THE NEW OUTBREAK SCENARIO FOR PUBLIC HEALTH ACTIVITIES

Our public health infrastructure is tiered, both in surveillance responsibilities and in response to emergency situations (39). At the local level, the county or city health department, first developed in response to epidemic cholera and other challenges in the 19th century, is responsible for most basic surveillance, investigation, and prevention activities. At the state level, epidemiologists, public health laboratorians, sanitarians, and educators conduct statewide surveillance and prevention activities and consult with and support local authorities. At the national level, CDC is the primary risk-assessment agency for public health hazards and conducts the primary national surveillance as well as epidemic response in support of state health departments. The Food and Drug Administration, Department of Agriculture, and Environmental Protection Agency are the primary regulatory agencies, charged with specific responsibilities regarding the nation's food and water supplies that interlock and are not always predictable. The Food and Drug Administration regulates low-acid canned foods, imported foods, pasteurized milk, many seafoods, rabbits raised for meat, and food and water provided on aircraft and trains. The Department of Agriculture regulates meat and poultry, including primary slaughter and further processing, and pasteurized **eggs**; investigates animal and plant diseases; and maintains the county extension outreach program. Shell **eggs** do not have a clear regulatory home, as the Department of Agriculture regulates the grading of shell **eggs** for quality, but the Food and Drug Administration, since 1995, has responsibility for the microbiologic safety of shell **eggs**.

The new outbreak scenario has several implications for the practice of public health, starting at the local level. One is that when diffuse outbreaks are detected, a local health department may need to investigate a few cases that are part of a larger outbreak despite their apparently small local impact. Second, an apparently local outbreak may herald the first recognized manifestation of a national or even international event.

When a diffuse outbreak of a potentially foodborne pathogen is detected, rapid investigation is needed to determine whether the outbreak is foodborne, and if possible, identify a specific food vehicle. These investigations, which typically include case-control studies, may need to be conducted in several locations at once. While all cases or all affected states may not need to be included in such an investigation, combining cases from several locations in one investigation and repeating the investigation in more than one location can be helpful. For example, in a recent international outbreak of Salmonella Stanley infections traced to alfalfa sprouts, concentrations of cases in Arizona, Michigan, and Finland led to case-control studies in each location, each of which linked illness to eating sprouts grown from the same batch of alfalfa seeds. This proved that the seeds were contaminated at the source (40). Parallel investigations can also lead to new twists. In the large West Coast outbreak of E. coli O157:H7 infections in 1993, a parallel investigation conducted in Nevada identified a type of hamburger other than the one implicated in the initial case-control investigation in Washington, leading to a broader recall and a more complete investigation of the circumstances of contamination (15,41). Because well-conducted investigations may lead to major product recalls, industrial review, and overhaul, and even international embargoes, it is essential that they be of the highest scientific quality.

Foodborne outbreaks are investigated for two main reasons. The first is to identify and control an ongoing source by emergency action: product

recall, restaurant closure, or other temporary but definitive solutions. The second reason is to learn how to prevent future similar outbreaks from occurring. In the long run this second purpose will have an even greater impact on public health than simply identifying and halting the outbreaks. Because all the answers are not available and existing regulations may not be sufficient to prevent outbreaks, the scientific investigation often requires a careful evaluation of the chain of production. This traceback is an integral part of the outbreak investigation. It is not a search for regulatory violations, but rather an effort to determine where and how contamination occurred. Often, the contamination scenario reveals that a critical point has been lost. Therefore, epidemiologists must participate in traceback investigations.

Intervention during outbreaks often depends on having enough good epidemiologic data to act with confidence, without waiting for a definitive laboratory test, particularly if potentially lethal illnesses are involved. For example, if five persons with classic clinical botulism ate at the same restaurant the preceding day (but have nothing else apparent in common), prudence dictates closing the restaurant quickly while the outbreak is sorted out--that is, before a specific food is identified or confirmatory cultures are made, which may take several days or even weeks. Good epidemiologic data, including evidence of a clear statistical association with a specific exposure, biologic plausibility of the illness syndrome, the potential hazard of that food, and the logical consistency of distribution of the suspect food and cases are essential.

The role of the regulatory agency laboratory is also affected by the new scenario. Because of the short shelf life and broad distribution of many of the new foods responsible for infection, by the time the outbreak is recognized and investigated the relevant food may no longer be available for culture. Because contamination may be restricted to a single production lot, blind sampling of similar foods that does not include the implicated lot can give a false sense of security. Good epidemiologic information pointing to contamination of a specific food or production lot should guide the microbiologic sampling and the interpretation of the results. Available methods may be insufficient to detect low-level contamination, even of well-established pathogens.

#### NEW APPROACHES TO THE PREVENTION OF FOODBORNE DISEASE

Meeting the complex challenge of foodborne disease prevention will require the collaboration of regulatory agencies and industry to make food safely and keep it safe throughout the industrial chain of production. Prevention can be "built in" to the industry by identifying and controlling the key points--from field, farm, or fishing ground to the dinner table--at which contamination can either occur or be eliminated. The general strategy known as Hazard Analysis and Critical Control Points (HACCP) replaces the strategy of final product inspection. Some simple control strategies are self-evident, once the reality of microbial contamination is recognized. For example, shipping fruit from Central America with clean ice or in closed refrigerator trucks, rather than with ice made from untreated river water, is common sense. Similarly, requiring oyster harvesters to use toilets with holding tanks on their oyster boats is an obvious way to reduce fecal contamination of shallow oyster beds. Pasteurization provides the extra barrier that will prevent *E. coli* O157:H7 and other pathogens from contaminating a large batch of freshly squeezed juice.

For many foodborne diseases, multiple choices for prevention are available, and the best answer may be to apply several steps simultaneously. For *E. coli* O157:H7 infections related to the cattle reservoir, pasteurizing milk and cooking meat thoroughly provide an important measure of protection but are insufficient by themselves. Options for better control include continued improvements in slaughter plant hygiene and control measures under HACCP, developing additives to cattle feed that alter the microbial growth either in the feed or in the bovine rumen to make cows less hospitable hosts for *E. coli* O157, **immunizing** or otherwise protecting the cows so that they do not become infected in the first place, and irradiating beef after slaughter. For *C. jejuni* infections

related to the poultry reservoir, future control options may include modification of the slaughter process to reduce contamination of chicken carcasses by bile or by water baths, freezing chicken carcasses to reduce *Campylobacter* counts, chlorinating the water that chickens drink to prevent them from getting infected, **vaccinating** chickens, and irradiating poultry carcasses after slaughter.

Outbreaks are often fertile sources of new research questions. Translating these questions into research agendas is an important part of the overall prevention effort. Applied research is needed to improve strategies of subtyping and surveillance. Veterinary and agricultural research on the farm is needed to answer the questions about whether and how a pathogen such as *E. coli* O157:H7 persists in the bovine reservoir, to establish the size and dynamics of a reservoir for this organism in wild deer, and to look at potential routes of contamination connecting animal manure and lettuce fields. More research is needed regarding foods defined as sources in large outbreaks to develop better control strategies and better barriers to contamination and microbial growth and to understand the behavior of new pathogens in specific foods. Research is also needed to improve the diagnosis, clinical management, and **treatment** of severe foodborne infections and to improve our understanding of the pathogenesis of new and emerging pathogens. To assess and evaluate potential prevention strategies, applied research is needed into the costs and potential benefits of each or of combinations.

To prepare for the 21st century, we will enhance our public health food safety infrastructure by adding new surveillance and subtyping strategies and strengthening the ability of public health practitioners to investigate and respond quickly. We need to encourage the prudent use of antibiotics in animal and human medicine to limit antimicrobial resistance. We need to continue basic and applied research into the microbes that cause foodborne disease and into the mechanisms by which they contaminate our foods and cause outbreaks and sporadic cases. Better understanding of foodborne pathogens is the foundation for new approaches to disease prevention and control.

Added material

Robert V. Tauxe Centers for Disease Control and Prevention, Atlanta, Georgia, USA

Address for correspondence: Robert V. Tauxe, Centers for Disease Control and Prevention, 1600 Clifton Road, MS A38, Atlanta, GA 30333 USA; fax: 404-639-2205.

Table 1. New pathogens that are foodborne and pathogens newly recognized as predominantly foodborne in the United States in the last 20 years

*Campylobacter jejuni*  
*Campylobacter fetus* ssp. *fetus*  
*Cryptosporidium parvum*  
*Escherichia coli* O157:H7 and related *E. coli*  
 (e.g., O111:NM, O104:H21)  
*Listeria monocytogenes*  
 Norwalk-like viruses  
*Nitzschia pungens* (cause of amnesic  
 shellfish poisoning)  
*Salmonella* Enteritidis  
*Salmonella* Typhimurium DT 104  
*Vibrio cholerae* O1  
*Vibrio vulnificus*  
*Vibrio parahaemolyticus*  
*Yersinia enterocolitica*

Table 2. Foodborne outbreaks traced to fresh produce, 1990-1996

Yr.	Pathogen	Vehicle	Cases		States
			(No.)	(No.)	Source
'90	S. Chester	Cantaloupe	245	30	C.A. (FNa)
'90	S. Javiana	Tomatoes	174	4	U.S. (FNb)

'90	Hepatitis A	Strawberries	18	2	U.S.
'91	S. Poona	Cantaloupe	>400	23	U.S./ C.A.
'93	E. coli O157:H7	Apple cider	23	1	U.S.
'93	S. Montevideo	Tomatoes	84	3	U.S.
'94	Shigella flexneri	Scallions	72	2	C.A.
'95	S. Stanley	Alfalfa sprouts	242	17	N.K. (FNC)
'95	S. Hartford	Orange juice	63	21	U.S.
'95	E. coli O157:H7	Leaf lettuce	70	1	U.S.
'96	E. coli O157:H7	Leaf lettuce	49	2	U.S.
'96	Cyclospora	Raspberries	978	20	C.A.
'96	E. coli O157:H7	Apple juice	71	3	U.S.

#### FOOTNOTES

a Central America

b United States

c Source not known

Table 3. Events and potential contamination sources during produce processing

Event	Contamination sources
Production and harvest	
Growing, picking, bundling	Irrigation water, manure, lack of field sanitation
Initial processing	
Washing, waxing, sorting, boxing	Wash water, handling
Distribution	
Trucking	Ice, dirty trucks
Final processing	
Slicing, squeezing, shredding, peeling	Wash water, handling, cross-contamination

Figure 1. Reported incidence of typhoid fever and nontyphoidal salmonellosis in the United States, 1920-1995.

Figure 2. Salmonella Enteritidis isolation rates from humans by region, United States, 1970-1996.

Figure 3. Incidence of three infections in FoodNet surveillance areas, 1996.

#### REFERENCES

1. Blake PA, Merson MH, Weaver RE, Hollis DG, Heublein PC. Disease caused by a marine vibrio: clinical characteristics and epidemiology. N Engl J Med 1979;300:1-5.
2. Riley LW, Remis RS, Helgeson SD, McGee HB, Wells JG, Davis BR, et al. Hemorrhagic colitis associated with a rare Escherichia coli serotype. N Engl J Med 1983;308:681.
3. Martin ML, Shipman LD, Wells JG, Potter ME, Hedberg K, Wachsmuth IK, et al. Isolation of Escherichia coli O157:H7 from dairy cattle associated with two cases of hemolytic uremic syndrome. Lancet 1986;2:1043.
4. Ortega YR, Sterling CR, Gilman RH, Cama VA, Diaz F. Cyclospora species--a new protozoan pathogen of humans. N Engl J Med 1993;328:1308-12.
5. Herwaldt BL, Ackers M-L, the Cyclospora Working Group. International outbreak of cyclosporiasis associated with imported raspberries. N Engl J Med. In press 1997.
6. Jackson LA, Wenger JD. Listeriosis: a foodborne disease. Infections in Medicine 1993;10:61-6.
7. Dekeyser PJ, Gossin-Detrain M, Butzler JP, Sternon J. Acute enteritis due to related Vibrio; first positive stool cultures. J Infect Dis 1972;125:390-2.
8. Tauxe RV. Epidemiology of Campylobacter jejuni infections in the

United States and other industrialized nations. In: Nachamkin I, Blaser MJ, Tompkins L, editors. *Campylobacter jejuni: current status and future trends*, eds. Washington (DC): American Society of Microbiology, 1992. pp 9-19.

9. Tauxe RV, Vandepitte J, Wauters G, Martin SM, Goosens V, DeMol P, et al. *Yersinia enterocolitica* infections and pork: the missing link. *Lancet* 1987;1:1129-32.

10. World Health Organization. Worldwide spread of infections with *Yersinia enterocolitica*. *WHO Chronicle* 1976;30:494-6.

11. Rodrigue DC, Tauxe RV, Rowe B. International increase in *Salmonella enteritidis*: a new pandemic? *Epidemiol Infect* 1990;105:21-7.

12. Centers for Disease Control and Prevention. Multidrug-resistant *Salmonella* serotype Typhimurium--United States, 1996. *MMWR Morb Mortal Wkly Rep* 1997;46:308-10.

13. Endt HP, Ruijs GJ, van Klingeren B, Jansen WH, van der Reyden T, Mouton RP. Quinolone resistance in *Campylobacter* isolated from man and poultry following the introduction of fluoroquinolones in veterinary medicine. *J Antimicrob Chemother* 1991;27:199-208.

14. Lee LA, Puhr ND, Maloney K, Bean NH, Tauxe RV. Increase in antimicrobial-resistant *Salmonella* infections in the United States, 1989-1990. *J Infect Dis* 1994;170:128-34.

15. Cieslak PR, Noble SJ, Maxson DJ, Empey LC, Ravenholt O, Legarza G, et al. Hamburger-associated *Escherichia coli* O157:H7 in Las Vegas: a hidden epidemic. *Am J Public Health* 1997;87:176-80.

16. Humphrey TJ, Greenwood M, Gilbert RJ, Rowe B, Chapman PA. The survival of salmonellas in shell **eggs** cooked under simulated domestic conditions. *Epidemiol Infect* 1989;103:35-45.

17. Kirkland KB, Meriwether RA, Leiss JK, MacKenzie WR. Steaming oysters does not prevent Norwalk-like gastroenteritis. *Public Health Rep* 1996;111:527-30.

18. Holmberg SD, Feldman RA. New and newer enteric pathogens: stages in our knowledge. *Am J Public Health* 1984;74:205-7.

19. St. Louis ME, Morse DL, Potter ME, DeMelfi TM, Guzewich JJ, Tauxe RV, et al. The emergence of Grade A **eggs** as a major source of *Salmonella enteritidis* infections: implications for the control of salmonellosis. *JAMA* 1988;259:2103-7.

20. Mishu B, J Koehler, Lee LA, Rodrigue D, Brenner FH, Blake P, Tauxe RV. Outbreaks of *Salmonella enteritidis* infections in the United States, 1985-1991. *J Infect Dis* 1994;169:547-52.

21. Besser RE, Lett SM, Weber JT, Doyle MP, Barrett TJ, Wells JG, Griffin PM. An outbreak of diarrhea and hemolytic uremic syndrome from *Escherichia coli* O157:H7 in fresh-pressed apple cider. *JAMA* 1993;269:2217-20.

22. Keene WE, Sazie E, Kok J, Rice DH, Hancock DD, Balan VK, et al. An outbreak of *Escherichia coli* O157:H7 infections traced to jerky made from deer meat. *JAMA* 1997;277:1229-31.

23. Kohn MA, Farley TA, Ando T, Curtis M, Wilson SA, Jin Q, et al. An outbreak of Norwalk virus gastroenteritis associated with eating raw oysters: implications for maintaining safe oyster beds. *JAMA* 1995;273:466-71.

24. Tauxe R, Kruse H, Hedberg C, Potter M, Madden J, Wachsmuth K. Microbial hazards and emerging issues associated with produce; a preliminary report to the National Advisory Committee on Microbiologic Criteria for Foods. *Journal of Food Protection*. In press 1997.

25. Wang G, Zhao T, Doyle MP. Fate of enterohemorrhagic *Escherichia coli* O157:H7 in bovine feces. *Appl Environ Microbiol* 1996;62:2567-70.

26. Zhuang R-Y, Beuchat LR, Angulo FJ. Fate of *Salmonella montevideo* on and in raw tomatoes as affected by temperature and **treatment** with chlorine. *Appl Environ Microbiol* 1995;61:2127-31.

27. Rushing JW, Angulo FJ, Beuchat LR. Implementation of a HACCP program in a commercial fresh-market tomato packinghouse: a model for the industry. *Dairy, Food and Environmental Sanitation* 1996;16:549-53.

28. Tauxe RV, Hughes JM. International investigations of outbreaks of foodborne disease: public health responds to the globalization of food. *BMJ* 1996;313:1093-4.



29. Hennessey TW, Hedberg CW, Slutsker L, White KE, Besser-Wiek JM, Moen ME, et al. A national outbreak of Salmonella enteritidis infections from ice cream. *N Engl J Med* 1996;334:1281-6.
30. Passarero DJ, Reporter R, Mascola L, Kilman L, Malcolm GB, Rolka H, et al. Epidemic Salmonella Enteritidis infection in Los Angeles County, California: the predominance of phage type 4. *West J Med* 1996;165:126-30.
31. Centers for Disease Control and Prevention. Outbreaks of Salmonella serotype Enteritidis infection associated with consumption of raw shell **eggs**--United States, 1994-1995. *MMWR Morb Mortal Wkly Rep* 1996;45:737-42.
32. Centers for Disease Control and Prevention. Summary of notifiable diseases, United States, 1995. *MMWR Morb Mortal Wkly Rep* 1995;44(53).
33. Centers for Disease Control and Prevention. Proceedings of a national conference on salmonellosis, March 11-13, 1964. U.S. Public Health Service Publication No 1262. Washington (DC): U.S. Government Printing Office; 1965.
34. Bean NH, Morris SM, Bradford H. PHLIS: an electronic system for reporting public health data from remote sites. *Am J Public Health* 1992;82:1273-6.
35. Bean NH, Goulding JS, Lao C, Angulo FJ. Surveillance for foodborne-disease outbreaks--United States, 1988-1992. *CDC Surveillance Summaries*, October 25, 1996. *MMWR Morb Mortal Wkly Rep* 1996;45(SS-5).
36. Centers for Disease Control and Prevention. Foodborne Diseases Active Surveillance Network, 1996. *MMWR Morb Mortal Wkly Rep* 1997;46:258-61.
37. Centers for Disease Control and Prevention. Establishment of a national surveillance program for antimicrobial resistance in Salmonella. *MMWR Morb Mortal Wkly Rep* 1996;45:110-1.
38. Hutwagner LC, Maloney EK, Bean NH, Slutsker L, Martin SM. Using laboratory-based surveillance data for prevention: an algorithm for detecting Salmonella outbreaks. *Emerg Infect Dis* 1997;3:395-400.
39. Meriwether RA. Blueprint for a national public health surveillance system for the 21st Century. *Journal of Public Health Management and Practice* 1996;2:16-23.
40. Mahon BE, Ponka A, Hall WN, Komatsu K, Dietrich SE, Siitonen A, et al. An international outbreak of Salmonella infections caused by alfalfa sprouts grown from contaminated seed. *J Infect Dis* 1997;175:876-82.
41. Bell BP, Goldoft M, Griffin PM, Davis MA, Gordon DC, Tarr PI, et al. A multistate outbreak of Escherichia coli O157:H7-associated bloody diarrhea and hemolytic uremic syndrome from hamburgers: the Washington experience. *JAMA* 1994;272:1349-53.

10/7/15 (Item 3 from file: 98)  
 DIALOG(R)File 98:General Sci Abs/Full-Text  
 (c) 2002 The HW Wilson Co. All rts. reserv.

03277841 H.W. WILSON RECORD NUMBER: BGS196027841 (THIS IS THE FULLTEXT)  
 Host specificity in microbe-insect interactions.  
 AUGMENTED TITLE: insect control by bacterial, fungal, and viral pathogens  
 Federici, Brian A  
 Maddox, Joseph V  
*BioScience* (BioScience) v. 46 (June '96) p. 410-21  
 LANGUAGE: English  
 COUNTRY OF PUBLICATION: United States  
 WORD COUNT: 11183

ABSTRACT: Part of a special section on biological pest control. A large number of pathogens that bring about disease in insects are among the alternatives to chemical insecticides. These pathogens generally have a far more narrow spectrum of activity than chemicals; as a result, the pathogens are extremely promising as selective biological control agents for insect pests. Only one insect pathogen, the bacterium *Bacillus thuringiensis*, is currently used frequently in the United States. The use of others, including a number of viruses, fungi, and protozoa, is likely to increase

as chemicals are used less frequently, however. The writers examine what is known about the host specificity of some of the most significant insect pathogens and discuss the probable consequences of expanding the use of these pathogens in biological control.

#### TEXT:

The need for alternatives to synthetic chemical insecticides has stimulated interest in developing more selective and environmentally sound insect control methods. The objective for many control programs is integrated pest management, which is a strategy wherein biological, chemical, genetic, and cultural controls are integrated, with chemicals being used only as a last resort.

Among the alternatives for chemical insecticides are many pathogens that cause diseases in insects. These pathogens typically have a much narrower spectrum of activity than do chemicals, and thus they have excellent promise for use as selective biological control agents for insect pests. Only one insect pathogen, the bacterium *Bacillus thuringiensis* (Bt), is used with any frequency at present in the United States. However, the use of others, including certain viruses, fungi, and protozoa, is expected to increase as the use of chemicals declines.

The prospect of using pathogens more widely as biological control agents is leading to greater scrutiny of their host specificity. Our current understanding of host specificity is derived from studies of pathogen biology as well as from empirical tests of host range. For most pathogens, including the more than 15 bacteria and viruses already registered for use as insect control agents, our knowledge of the biological mechanisms underlying host specificity remain largely unknown. Decisions regarding use have therefore been based primarily on the results of empirical laboratory tests. The extent to which pathogens will be used in the future will depend in part on knowledge about host specificity. Interpretation of this knowledge by governmental agencies must be based on sound biological principles.

In this article, we examine what is known about the host specificity of some of the most important insect pathogens, and, based on this knowledge, we draw some conclusions about the likely consequences of expanding their use in biological control. Two of the most important questions are: What is the likelihood that the host range of an insect pathogen can expand to include nontarget hosts not currently affected? How will exotic (nonindigenous) pathogens affect native nontarget hosts? Insect pathogens may affect nontarget hosts by causing direct and relatively immediate mortality. This concern is the most important one for pathogens used as insecticides, in which the pathogen is applied at a high rate in comparison to the amount normally present in a habitat. A pathogen may also colonize a nontarget insect host population and cycle indefinitely in that population. This situation is more a concern with exotic pathogens introduced into the United States.

To place the issue of host specificity in perspective, we provide a few examples of the natural occurrence of insect pathogens in the environment. We then describe strategies for using different types of pathogens as insect control agents. Finally, we examine the issues and complexities of host specificity for the different types of insect pathogens.

#### INSECT DISEASES

Most insect species can be afflicted by an assortment of infectious diseases caused by viruses, bacteria, fungi, and protozoa. Many of these entomopathogens are widely distributed in the environment and can be readily isolated from soil, water, plant surfaces, air, and excretions and bodies of infected insect hosts. Viral and protozoan pathogens persist at low levels in the insect population and spread slowly through it before a disease outbreak occurs. This spread of the pathogen usually occurs as host population density increases. Fungi more commonly persist as resistant spores in the environment, with disease outbreaks being triggered by special conditions of temperature, humidity, and population density. Bt does not cause disease outbreaks but occurs commonly in soil and other

substrates around the world.

In many insect species, disease outbreaks occur routinely and serve as natural regulators of insect populations (Tanada and Fuxa 1987). Pathogens causing such disease outbreaks in insects include the nuclear polyhedrosis viruses of forest pests such as the gypsy moth, *Lymantria dispar*, and the Douglas fir tussock moth, *Orgyia pseudotsugata*, and many fungal entomopathogens that occur seasonally in grasshopper, fly, and aphid populations.

Less noticeable, but equally effective, are the population reductions resulting from pathogens that cause less acute diseases. Protozoan diseases, for example, in populations of the European corn borer, *Ostrinia nubilalis*, and in Eurasian populations of the gypsy moth, *L. dispar*, are effective in reducing the magnitude of pest infestation.

When disease outbreaks occur, the pathogen load in the environment may be high, even if only for a few days. During such outbreaks, many nontarget organisms, including other insects, and vertebrates, including humans, are periodically exposed to insect pathogens.

Given that insect pathogens are so numerous and widespread in the environment and that they periodically cause disease outbreaks in many pest populations, it is reasonable to ask why it is necessary to add additional pathogens to control pest populations. The answer is because insect pest populations generally exceed their economic threshold--the population at which significant economic damage results--before a disease outbreak occurs. Consequently, the goal of introducing insect pathogens into pest populations is to induce disease outbreaks before the population reaches an economic threshold.

#### HOW ARE INSECT PATHOGENS USED?

The marked declines in insect populations that follow disease outbreaks led researchers to suggest more than 100 years ago that the pathogens might be used to control insect pests (Cameron 1973). This idea has received serious study over the past 50 years, and four different tactics for using pathogens for insect control have emerged: classical biological control, augmentative releases, inundative releases, and environmental manipulation.

Classical biological control is the ideal tactic. It is permanent and requires little or no intervention. In classical biological control, a pathogen (usually nonindigenous) is introduced into a pest population at one or a few geographical locations. The pathogen recycles in the target pest population and becomes endemic within a few years, permanently reducing the pest infestation to a lower level and then maintaining this level.

An excellent example of classical biological control is the use of the nuclear polyhedrosis virus of the European spruce sawfly, *Gilpinia hercyniae*. The sawfly invaded Canada and the United States from northern Europe during the last century, and by early in this century had become a major pest of spruce forests. The virus was introduced from Sweden in the 1930s, apparently with virus-contaminated parasitic wasps released for biological control. By 1943 the virus had reestablished a natural balance with the spruce sawfly (Balch and Bird 1944), reducing the pest population below an economic threshold, where it remains today. Other classical biological control agents include *Nosema pyrausta*, a microsporidium of the European corn borer, *O. nubilalis*, and *Entomophaga maimaiga*, a fungal pathogen of the gypsy moth, *L. dispar*. Although these pathogens do not keep their hosts below an economic threshold on an ongoing basis, they periodically cause outbreaks of disease that prevent significant economic loss.

A second tactic is augmentative release. In this case, pathogens are periodically added to populations in which they already occur to increase the pathogen load. The result is that a disease outbreak is initiated before it would otherwise have occurred, thereby reducing the size of the pest population below the economic threshold. With this approach, a single introduction or inoculative release cannot be relied on to give ongoing control, and additional pathogens must be added, either after several years

or, in some cases, on a seasonal basis.

An excellent example is the use of the bacterium *Bacillus popilliae* to control the Japanese beetle, *Popillia japonica*, the larvae of which are important pests of grasslands and turf. This bacterium rarely causes disease outbreaks under natural conditions. However, when applied as an augmentative release, a single application may provide effective pest control for up to ten years (Klein 1981). Another example is use of the microsporidium *Nosema locustae* to reduce grasshopper populations. This pathogen is introduced periodically to populations on a corn cob bait, on which the grasshoppers feed, as an augmentative release (Henry and Onsager 1982).

The most common tactic for controlling insects with pathogens is to make an inundative release of the pathogen into a pest population with the expectation of relatively quick host mortality. The term microbial insecticide describes pathogens used in this way. The pathogen is applied as needed to keep the pest population below the economic threshold. Typically, this tactic requires fewer applications than a broad-spectrum chemical pesticide would because the pathogen is much more host specific than most chemicals and thus preserves other natural regulators of the pest (e.g., spiders and predatory and parasitic insects). The tactic of developing pathogens as microbial insecticides has received the most attention because it has proven the most successful for controlling pests and for generating profits for the pesticide industry.

Environmental manipulation is a tactic in which the effectiveness of insect pathogens that are already present is improved on. This approach is more often used in the practice of organic farming and sustainable agriculture than in intensive agricultural systems. Nevertheless, manipulating cropping practices (irrigation, harvesting, cultivation practices, and row spacing) can affect the onset and intensity of disease outbreaks. Information currently available on these practices is insufficient to determine whether or how manipulation of the environment is likely to affect host specificity of insect pathogens.

#### TYPES OF INSECT PATHOGENS

Our knowledge of host specificity varies with pathogen type, and some types are used to control insect pests much more commonly than are others. Thus, we discuss each group separately. For each pathogen type, we illustrate key points using one or two pathogen species, with those discussed being selected on the basis of their level of current use or that envisioned for the near future.

A good representative for bacterial pathogens of insects is the common microbial insecticide, Bt. Of all insect pathogens, we know the most about the determinants of host specificity for Bt. For the viruses, the representatives we discuss are the nuclear polyhedrosis viruses of lepidopterous insect pests, because several of these are already registered as microbial insecticides, and because they are the best studied. For the fungi, we discuss both *Beauveria bassiana* (Bb), a ubiquitous complex of strains isolated from many insect species, and *Zoopthora phytonomi*, a much more fastidious species. Finally, for the protozoa we discuss the microsporidium *Nosema algerae*, a pathogen of mosquitoes, and *Vairimorpha necatrix*, a pathogen of lepidopteran larvae.

#### BACTERIA

The bacterium Bt is by far the pathogen used most commonly in insect control in the United States, with thousands of tons of Bt applied annually to vegetable and field crops, ornamentals, and forests to control caterpillar and beetle pests. Bt is also used in aquatic habitats to control mosquito larvae. Bt is common in the environment and is easily isolated from soil as well as from other sources, including insects, insect feces, grain dust, and the leaves of deciduous plants (Meadows 1993). Little is known about its ecology despite its widespread use in pest control.

Bt is a spore-forming bacterium that during sporulation produces a

crystalline parasporal body composed of one or more insecticidal proteins (Figure 1). The spore itself contributes to insecticidal activity when Bt is used against some insect species, but most pests are killed by the parasporal body proteins. In either case, Bt must be eaten by an insect to be toxic. Once within the insect midgut, the protein crystals dissolve, and the insecticidal parasporal proteins are activated by midgut protease enzymes (Pietrantonio et al. 1993). The activated toxin or toxins bind to specific protein receptors on midgut cells of susceptible insects, causing these cells to lyse and the insect to die. For most insects, toxin activation occurs under alkaline conditions, usually when the pH is 8 or higher. A key aspect of host specificity for Bt is the recognition of specific binding sites (receptors) on insect midgut cells by the activated toxins. Although crystals dissolve in many insects with alkaline midguts and are activated by midgut proteases, the activated toxins will not be toxic in the absence of midgut binding sites.

Bt is not a single organism but rather a complex of more than 50 subspecies that can be distinguished on the basis of immunological characteristics (H antigen serotype) of flagellar proteins on the cell surface (de Barjac and Franchon 1990). The genes that encode the insecticidal proteins are typically located on large transmissible plasmids. The host specificity of any Bt isolate is determined by these plasmids, which encode the complement of insecticidal proteins produced during sporulation and assembled into the parasporal body. Different isolates of the same subspecies can have different plasmid complements and, therefore, can differ in their host specificity.

Most of the insecticidal proteins are referred to as Cry (for crystal) proteins and are classified on the basis of their activity spectrum and molecular mass (Hofte and Whiteley 1989). For example, CryI proteins are active against the larvae of lepidopterous insects and have a mass of approximately 135 kilodaltons (kDa), whereas CryII proteins are also active against lepidopterous insects but have a mass of approximately 65 kDa. The 65-kilodalton CryIII proteins are toxic to coleopterous insects, such as the Colorado potato beetle, *Leptinotarsa decemlineata*. CryIV proteins are toxic to the larvae of certain dipterous insects, such as mosquito and blackfly larvae, and can be in the range of either 135 or 65 kDa. Within each general type of Cry protein, many natural variants occur that can differ greatly in their specific toxicity against a particular insect species (Table 1). All Cry proteins share significant homology at the amino acid sequence level, and it is likely that they evolved from a single ancestral protein. The Cry proteins of 65 kDa are truncated versions of the 135-kilodalton proteins that correspond to their N-terminal portions. Although mixtures of different Cry protein types can occur in the same Bt isolate, the overwhelming majority of isolates evaluated to date are active only against the insects of a single order, that is, dipterans, coleopterans, or lepidopterans.

Proteolytic activation of Cry proteins in the insect midgut results in a toxin of approximately 60 kDa. Comparison of more than 50 Cry protein genes has revealed that these toxins contain both highly conserved and highly variable blocks of amino acids. Recombinant DNA experiments and x-ray crystallographic studies indicate that the toxin domain is located primarily in the N-terminal half of the activated Cry molecule, with the specificity domain being localized primarily in the variable blocks of amino acids on the C-terminal half (Ge et al. 1989, Hofte and Whiteley 1989, Li et al. 1991). The variable blocks of amino acids enable the toxin to bind to specific glycoprotein receptors on the midgut microvillar membranes of susceptible insects (Sangadala et al. 1994). Subsequently, the toxin inserts into the cell membrane, forming pores that cause cell lysis, and eventually, death of the insect. Thus, the spectrum of activity of a particular Cry protein is determined by specific amino acid sequences located in the C-terminal half of the activated protein.

Although the precise molecular mode of action of Bt toxins is not yet understood, our knowledge of the molecular genetics of Cry proteins explains the activity and host specificity of many isolates, including those used in commercial strains. If a Bt isolate produces more than one Cry protein, as many do, it is likely to have a broader spectrum of

activity than an isolate that produces only one Cry protein. For example, the HD1 isolate of *B. thuringiensis* subsp. *kurstaki*, the isolate used in many commercial formulations of Bt (Table 2), is more toxic to and has a broader spectrum of activity against lepidopterous insects than the HD73 isolate of the same subspecies. Both produce large, bipyramidal crystals; the HD1 isolate also produces a much smaller, cuboidal crystal (Figure 1b). Genetic and immunological analyses of the parasporal inclusions produced by these two isolates show that the plasmids carried by the HD73 isolate expresses only the gene for the CryIA(c) protein. The bipyramidal crystal produced by this isolate is composed solely of this protein. The HD1 isolate carries a different complement of plasmids, and from these, four genes are expressed, those encoding the CryIA(a), CryIA(b), CryIA(c), and CryIIA proteins. The three CryIA proteins cocrystallize to form the bipyramidal crystal, and the CryIIA protein crystallizes separately to form the smaller cuboidal parasporal body. This isolate also carries a gene for the CryIIB protein, but this gene is not expressed.

During the 1980s, each of these genes was cloned and expressed, and the resultant proteins were tested for specific toxicity and spectrum of activity. As shown in Table 1, the CryIA(c) protein is highly toxic to some lepidopteran species, such as the tobacco budworm, *Heliothis virescens*, but virtually nontoxic to the cabbage worm, *Mamestra brassicae*. By contrast, the CryIA(a) protein is highly toxic to the tobacco hornworm, *Manduca sexta*, and moderately toxic to *H. virescens* and *M. brassicae*. A comparison of the total spectrum of activity of the four proteins that occur in HD1 with the single protein that occurs in HD73 makes apparent the reason for the difference in the spectrum of activity: The sum of the spectrum of activity of each of the Cry proteins produced by HD1 is greater than the spectrum of activity of the single Cry protein in HD73 (Table 1).

It is important to note that when the first formulations of HD1 were registered by the US Environmental Protection Agency (EPA) more than 30 years ago, nothing was known about the molecular genetics of Bt. Information was obtained about host specificity by empirical testing. These methods continue to be used today for new isolates of Bt, as well as for other insect pathogens being registered for use as microbial insecticides. This is because our current knowledge of Bt is insufficient to allow us to predict the host specificity of new isolates.

The protocols for determining the degree of host specificity and safety are established by EPA (Betz et al. 1990, Lacey and Mulla 1990). These involve extensive testing of Bt against a range of target and nontarget insects and nontarget vertebrate species, such as rodents and birds. Bt preparations are evaluated in vertebrates for such properties as dermal, oral, and intravenous toxicity, teratogenicity, and carcinogenicity. They are also evaluated for oral and contact toxicity against beneficial insects, including honeybees, parasitic wasps, and predatory insects and spiders. Bt strains are registered only after a substantial amount of evidence has been provided to EPA showing that no undesirable effects have been obtained at doses ranging up to 1000-fold the amount of the preparation that would be applied as an insecticide.

The tests required for registration do not answer questions about potential shifts in host specificity, however. In this context, the time scale being considered is important. If all Cry proteins evolved from a single ancestral protein, clearly the host specificity can evolve and diversify. The use of Bt will probably continue to expand over a period of perhaps another 50-100 years, until other pest control technologies replace microbial insecticides. Mutations will occur in Bt genes, and because tons of Bt are being produced and released into the environment, there is significant opportunity for the genetic diversity of the strains in the products being applied to increase. But even with increased usage, it is highly unlikely that through random mutation a larger variety of cry genes will be generated than already have evolved and occur naturally.

Our current knowledge of the genetic diversity of Cry proteins suggests that those mutations that lead to host shifts are more likely to lead to shifts within host groups (e.g., lepidopteran to lepidopteran) than between host groups (e.g., lepidopteran to coleopteran). Interclass or

interphyla shifts are even less likely. Thus, no evidence exists indicating that current or increased use of Bt will substantially shift or expand the spectrum of activity of this bacterium.

Another concern that must be addressed in regard to the host specificity of existing Bt isolates used in commercial preparations is the effect on nontarget species, especially endangered species. For example, what will happen to nontarget species of lepidopterous insects that are exposed to Bt in the course of **treating** large forests, as is done in the case of the gypsy moth? Because most commercial preparations of Bt used against lepidopterans are based on the "broad-spectrum" HD1 isolate, which is active against many species of lepidopterous pests, nontarget lepidopterous insects are likely to suffer significant mortality in **treated** areas (Miller 1990). However, the impact of Bt would still be less than that of broad-spectrum chemical insecticides, which kill many nontarget arthropods, or than no **treatment** at all. The latter would allow significant crop destruction, and perhaps even greater impact on nontarget endangered species because of the loss of habitat.

#### VIRUSES

Nuclear polyhedrosis viruses (NPVs) are the viruses most commonly considered for use as insect control agents (Federici 1993). This use is directed almost exclusively against lepidopterous pests. NPVs do not generally work well as classical biological control agents or when used in augmentative release programs because even when large amounts of virus are applied the level of insect control is inadequate. Thus, they are viewed primarily as microbial insecticides, to be used as needed in integrated pest management programs. Eight viruses, seven of which are NPVs, are presently registered by EPA for use as insecticides, but only the gypsy moth NPV is in current use. Its use, sponsored by the US Forest Service, amounts to less than 8000 acres **treated** per year. No viral insecticides are currently sold commercially in the United States. Until recently, chemical insecticides and Bt products have been available to control the same caterpillar pests against which the NPVs are targeted. However, this situation may soon change because of the observed resistance to chemical insecticides in many lepidopteran pests, the potential for the development of resistance to Bt, and renewed interest on the part of the pesticide industry to developing alternatives to chemical insecticides.

NPVs are large, complex, double-stranded DNA viruses characterized by the production of enveloped rod-shaped virions. The genome is large (130 kbp) and capable of encoding at least 100 proteins. Viral replication and virion assembly occur in the nuclei of infected tissues; once formed, virions are occluded in angular protein crystals referred to as polyhedra, or occlusion bodies (Figure 2). Several hundred NPV isolates have been collected over the past 50 years, the majority from lepidopterous species (Martignoni and Iwai 1986a). More than 15,000 species of Lepidoptera occur in the United States, and it is probable that each is afflicted periodically by one or more NPV diseases. NPVs have also been isolated from insects belonging to the orders Diptera (flies), Trichoptera (caddis flies), Thysanura (silverfish), Hymenoptera (sawflies), Coleoptera (beetles), Siphonaptera (fleas), as well as from shrimp (e.g., *Penaeus monodon*), but they are much less common in these groups.

Insects typically become infected by NPVs when they ingest polyhedra on foliage. The polyhedra dissolve in the midgut, and the released virions invade the insect through the midgut epithelium microvilli. An initial phase of replication occurs in midgut epithelium nuclei, after which progeny virions invade most other tissues in the body, leading to the formation of infectious polyhedra. In insect species with low susceptibility to a particular NPV, virus replication may be restricted to the midgut epithelium and to only a small number of cells in other tissues. Members of insect species so infected usually survive infection and are capable of continued growth, development, and reproduction.

As with Bt, little is known about the ecology of NPVs, but many periodically cause widespread disease outbreaks that bring about rapid declines in populations of lepidopterous larvae. These outbreaks of

disease occur in populations that reach densities of several thousand to several hundred thousand larvae per acre. Depending on the species afflicted, these outbreaks may occur on rangeland, forests, or large plantings of vegetable and field crops. During such disease outbreaks, pounds of virus per acre can be released into the environment and come in contact with, and be ingested by, many other organisms, including other insects, birds, rodents, and humans (Tanada and Fuxa 1987, Thomas et al. 1974).

The molecular determinants of host range in NPVs remain largely unknown. Most of our information about host specificity has come from empirical studies, which have shown that individual viruses differ considerably in their degree of host specificity (Groner 1990). A few, such as the alfalfa looper (*Autographa californica*) NPV (AcNPV) have a broad host range within the order Lepidoptera. AcNPV is known to infect and reproduce in at least 33 lepidopteran species belonging to ten families. The infectivity and virulence of this virus varies among different species and has not been well characterized. AcNPV may also be capable of infecting and replicating in insects of other orders, but this ability has not been demonstrated conclusively. Despite the broad host spectrum determined in laboratory studies, AcNPV has been collected in the field only from species of the lepidopteran family Noctuidae. Other NPVs, such as the NPV of the beet armyworm, *Spodoptera exigua*, are highly specific in laboratory studies. Laboratory host specificity experiments with this virus indicate that it can replicate efficiently only in *S. exigua*. Other NPVs fall between these two extremes and are capable of infecting several closely related lepidopteran species. An example of an NPV with this fairly restricted host spectrum is the *Heliothis* NPV (HzNPV), which is capable of infecting several species of *Heliothis* and *Helicoverpa* (Ignoffo 1975). HzNPV is one of the viruses registered as a microbial insecticide.

Until recently, virtually nothing was known about the genetic basis for host specificity in NPVs or other insect viruses. Maeda et al. (1993) have now shown that a recombinant NPV containing portions of AcNPV and portions of the *Bombyx mori* NPV (BmNPV) can infect *B. mori* cells, something that AcNPV cannot do. Genetic analysis of the recombinant virus showed that the expanded host range was made possible by the introduction of the BmNPV helicase (an enzyme involved in DNA replication) gene into the AcNPV genome. Similar results have also been reported by Croizier et al. (1994). Because these two viruses are closely related, the difference in their host specificity may be due to a single gene. The complexity of NPVs indicates that, as with nearly all viruses, successful NPV reproduction in a particular host is dependent on the coordinated expression of several viral and host genes. When a virus winds up in a host outside its normal host range, this coordination is less than optimal. The more distant the relationship between the virus host and its normal host(s), the less likely it is that viral reproduction will be successful. This complexity likely accounts for the high degree of host specificity observed for most NPVs in the laboratory and field, although the molecular details remain to be elucidated.

Although our understanding of the molecular mechanisms underlying NPV host specificity is in its early stages, laboratory studies as well as the phylogenetic distribution of NPVs enable us to make a prediction about potential shifts or expansions in host range. We predict that although individual NPVs may be capable of shifting or expanding host range within a related group (e.g., among species of the same order), more distant host shifts or expansions are highly unlikely. Laboratory studies have already demonstrated that cross-family host shifts are possible within the order Lepidoptera. For example, Martignoni and Iwai (1986b) showed that the host specificity of the NPV of the Douglas fir tussock moth, *O. pseudotsugata* (family Lymantriidae), could be shifted to include the cabbage looper, *Trichoplusia ni* (family Noctuidae). They accomplished this host shift by feeding cabbage looper larvae large doses of the tussock moth NPV over a period of 12 sequential generations. During the early generations mortality was low, and histological analysis showed only a low level of infection in most tissues. However, over successive generations, the level



of infection in tissues increased. By the seventh generation, the virus had completely adapted to the cabbage looper. Interestingly, the passaged virus also increased its virulence for its original host, the tussock moth, by tenfold.

This study makes it clear that NPVs can undergo host shifts. Although forced feeding does not represent natural selective conditions (with the possible exception of NPV disease outbreaks in dense populations), repeated use of a viral insecticide in the same area could result in much higher levels of exposure to virus in nontarget populations than occurs normally. With viruses that are quite host specific, such as the beet armyworm (*S. exigua*) or corn earworm (*Helioverpa zea*) NPVs, host shifts are unlikely. However, with viruses of less restricted host specificity, such as AcNPV, it is more difficult to rule out the possibility of shifts or expansions in host range.

Other studies provide strong evidence that broader shifts in host range are unlikely to occur. For example, several NPVs from lepidopterous insects have been tested extensively against insects from other orders and vertebrates, but none has ever been shown to be capable of reproduction in these nontarget hosts (Groner 1986, 1990). For a shift to occur, there must be at least some reproduction in the nontarget host. Although mutations might arise that would make this possible, given the complexity of NPVs a host shift is unlikely because it would probably require tens, if not hundreds, of simultaneous mutations. If a major shift or expansion were to occur, it would first be to insects of another order, not to members of another class or phylum. Such significant shifts are possible through gradual change over evolutionary time. Assuming that NPVs originated from a common ancestor, their phylogenetic distribution shows that such shifts have occurred. Yet these shifts occurred over millions of years, and, as noted for Bt, the upcoming window of opportunity for the use of viruses as insect control agents is likely to be less than 100 years.

#### FUNGI

More than 50 genera of fungi contain species that are pathogenic to insects (McCoy et al. 1988). These species have extremely heterogeneous life cycles, produce assorted pathologies in their infected hosts, and exhibit varying degrees of host specificity. Most fungal pathogens of insects are transmitted from infected hosts to susceptible hosts by environmentally resistant forms known as spores (sexual forms) or conidia (asexual forms). In the typical fungal life cycle, when a spore or conidium (Figure 3) encounters a susceptible host, it adheres to the cuticle, germinates, and penetrates the body wall of the host by means of its germ tube. Inside the host, the fungus grows vegetatively, often as yeastlike hyphal bodies. The host usually dies, after which the fungus forms typical mycelia that grow out through the cuticle and produce more resistant conidia. These conidia may then infect other hosts. The life cycles of many insect pathogenic fungi are more complicated than those of typical fungi and may involve alternate hosts, motile spores, and the production of toxins. Host specificity may be limited if any one of these events is circumvented. The details of fungal life cycles and pathogenesis are reviewed by Goettel et al. (1990), McCoy et al. (1988), and Samson et al. (1988).

Most studies of host specificity in entomopathogenic fungi have dealt with interactions between the conidium and the insect cuticle. For several fungal species, it has been demonstrated experimentally that attachment of the conidium to the cuticle and penetration through the cuticle and body wall are greatly influenced by the chemical composition of the cuticle (Boucias and Latge, 1988, Boucias and Pendland 1984, Boucias et al. 1988, St. Leger 1993). The cuticle is clearly a major factor that determines the host specificity of many fungal pathogens. Cellular and chemical immune responses occur when some fungi invade the hemocoel of nontarget hosts. These responses, although not well documented, affect host specificity. We discuss two species of entomopathogenic fungi to illustrate some of the host specificity characteristics of fungal entomopathogens.

*Beauveria bassiana* (Bb) is a common but little understood fungal pathogen of many groups of insects. Bb is a member of the Deuteromycotina

(Fungi Imperfecti), an assemblage of genera that reproduce asexually only. The life cycle is relatively simple. A conidium attaches to a host, germinates, and forms a germ tube that penetrates the cuticle and body wall. The fungus then grows vegetatively inside the host body. The host dies, and the fungus grows through the body wall and produces more conidia. Bb has been used for many years as a microbial insecticide in the former Soviet Union and in the People's Republic of China (Ferron 1981, McCoy et al. 1988). It grows saprophytically on artificial media and can be mass produced in quantities sufficient to **treat** thousands of acres.

Bb has been reported to infect more than 100 insect species in many different insect orders (McCoy et al. 1988). However, individual isolates of Bb from a particular insect species, when tested against a range of insect species in the laboratory, have a narrow host range, and thus each may be relatively host specific (Fargues 1976, Ferron et al. 1972). The host specificity of some isolates is related to cuticular differences among different insect host species. Additional factors have been identified that may further restrict host range in nature. For example, Fargues and Remaudiere (1977) found that under field conditions, spatial and temporal factors influence the host specificity of some groups of fungi. And Ramoska and Todd (1985) found that different host plants affect the susceptibility of the cinch bug, *Blissus* species, to infection by Bb.

The dilemma in estimating the host specificity of Bb is similar to the one that arises with many other insect pathogens. Fungi identified as Bb have been isolated from many different insect species representing many orders. Conventional morphological taxonomic characters are not sufficiently different to distinguish between these isolates, even though different isolates (pathotypes) of Bb are often relatively host specific, even in laboratory studies. Molecular approaches to the identification of pathotypes are being developed and should help to resolve some of these problems.

In comparison to the broad host range of the typical deuteromycete fungus, species of entomophthoraceous fungi (division Zygomycotina) are usually much more specific. Even so, there are problems with their identification and determination of their host specificity. *Z. phytonomi*, a pathogen of weevil larvae in the genus *Hypera* (order Coleoptera), was first reported in North America in 1973 in Ontario, Canada, attacking the alfalfa weevil, *Hypera postica* (Harcourt et al. 1974). The alfalfa weevil is of European origin and was accidentally introduced near Salt Lake City, Utah, in 1904. Over the past 90 years, it has gradually extended its range to include all contiguous 48 states (Steffey et al. 1994). A fungus similar to *Z. phytonomi* occurs in alfalfa weevil populations in Europe. Another weevil, the clover leaf weevil, *Hypera punctata*, is native to North America and is attacked routinely by a fungus that is similar to *Z. phytonomi*. The latter fungus is an important naturally occurring biological control agent of the clover leaf weevil.

The two weevil species were sympatric in different regions of North America for several years before the report of *Z. phytonomi* in Canadian populations of the alfalfa weevil. In addition, since that report, *Z. phytonomi* infections in alfalfa weevils have spread outward from the location of the initial siting in Canada, and the fungus is now present throughout North America. Is the *Z. phytonomi* that infects the alfalfa weevils a fungus introduced from Europe, or did the fungus in the clover leaf weevil adapt to the alfalfa weevil and expand its host range? If the host range of the fungus in the clover leaf weevil expanded, it required several years, and it probably occurred in a single location.

More significant host range expansions are unlikely to occur. Little has been published on laboratory host range experiments, but our preliminary experiments indicate that *Z. phytonomi* from the alfalfa weevil will infect few, if any, hosts outside the genus *Hypera*.

#### PROTOZOA

The Protozoa are a large and heterogeneous group of single-celled organisms that possess nuclei. Most taxonomists now recognize Protozoa as a subkingdom with at least seven phyla; five of these phyla contain species that are pathogenic to insects (Brooks 1988, Canning 1990). We focus on

the phylum Microspora, which, from the standpoint of entomopathogens, is the most important. Only one microsporidium, *N. locustae*, a pathogen of grasshoppers, has been registered as a microbial insecticide. Its use is limited to specific rangeland situations. Because it kills grasshoppers slowly, it is doubtful that *N. locustae*, or any other microsporidium, will ever be used extensively as a microbial insecticide: the diseases they cause are chronic rather than acute (Canning 1982). Microsporidia periodically build up in many pest populations, leading to disease outbreaks that regulate or control these pests. Though so far they have been used deliberately only rarely in pest control, microsporidia hold considerable potential for use as classical biological control agents.

Microsporidia are obligate intracellular pathogens transmitted from one host to another by the ingestion of small resistant spores. When eaten by a susceptible host, the spore extrudes a unique organelle called a polar filament (Figure 4). The sporoplasm is expelled from the spore through the hollow polar filament and **injected** into the midgut cells of the host. Inside these cells, the microsporidia multiply vegetatively, often with complex life cycles. The pathogen forms more spores, which are released into the environment in feces, silk, or when the host dies. They may, depending on the species, infect various tissues, have alternate hosts, have sexual cycles, or produce several types of spore. Some species of microsporidia may be transmitted vertically from infected females to their progeny: Spores may occur on the **egg** surface, or various forms of the microsporidium may occur inside the **egg** in the form of an active infection. There is no "typical" microsporidian infection because the group is so heterogeneous.

The specific conditions (pH and ionic concentrations) in the stomach of the host have an important influence on host specificity (Undeen and Maddox 1973). If spores are not stimulated to extrude their contents in the midgut, infection cannot progress. Cellular immune responses of insects to microsporidian infections have been documented for many species of microsporidia (Brooks 1988). Such immune responses probably influence host specificity. We discuss the characteristics of two microsporidian pathogens of insects as examples of host specificity in the microsporidia.

*N. algerae* is a microsporidian pathogen of mosquitoes. It has been experimentally applied as both a microbial insecticide and as an augmentative biocontrol agent for the control of several mosquito species. It is not presently being considered for commercial development, and it is unlikely to be so considered in the future, because it is too expensive to produce in the amounts needed to achieve effective mosquito control. In the laboratory, *N. algerae* has an extremely wide host range. It infects many, although not all, mosquito species (Maddox et al. 1981, Savage 1975). It can also be transmitted to two muscoid flies, four species of Coleoptera, six species of Lepidoptera, one species of Hemiptera, and two species of trematodes (Brooks 1988). When infectious forms of *N. algerae* were **injected** directly into nontarget hosts (bypassing the need for the spores to extrude in the midgut), six orders of insects, a crayfish, and white mice became infected (Undeen and Maddox 1973). Most of the experimental infections were abnormal, and it is highly unlikely that the pathogen could cycle in those nontarget hosts. In addition, *N. algerae* infections have never been found in field-collected hosts other than anopheline mosquitoes (Brooks 1988).

*V. necatrix* is a microsporidian pathogen of lepidopterous insects. It is one of the most pathogenic microsporidian species known. Although it has been investigated as a potential microbial insecticide and many experimental field applications have been made, it is doubtful that it will ever be available commercially as a microbial insecticide because spores must be grown in larvae and the costs are too high considering the number of larvae that must be produced to obtain effective pest control.

Field-collected isolates of *V. necatrix* have identical life cycle and ultrastructural characteristics, but they exhibit different host specificity under laboratory conditions. This species infects a wide range of nontarget lepidopterous species if large doses of spores are fed to larvae (Maddox et al. 1981). However, when 20 different isolates from field-collected lepidopteran larvae were fed to several nontarget hosts in

the laboratory, some isolates produced abnormal infections, in which atypical tissues were infected, abnormal spores were produced, and infections were sometimes uncharacteristically light. Therefore, *V. necatrix*, like *Bb*, has pathotypes that vary in host range, but these cannot be distinguished from one another using conventional taxonomic characters. At present, we know virtually nothing about the determinants of host specificity for microsporidia. As with other insect pathogens, host range has been determined by empirical tests in the laboratory (Betz et al. 1990). However, because microsporidia are not being considered for use as microbial insecticides, few have been evaluated for host range using these tests.

#### RECOMBINANT PATHOGENS

Pathogens are emerging as useful components of insect control programs, but their relatively high degree of host specificity and slow speed of kill in comparison to broad-spectrum chemical insecticides are often cited as impediments to further development. To overcome these disadvantages, genetic and recombinant DNA techniques are being used to increase the efficacy of pathogens by increasing their host range and virulence.

For example, *Bt* plasmids from different isolates with different target insect spectra have been combined in a single strain to produce a novel *Bt* with an expanded spectrum of activity. This strategy was used by Ecogen, Inc., of Langhorne, Pennsylvania, to produce the commercial product Foil(r). This strain of *Bt* subsp. *kurstaki* contains two plasmids, one bearing genes that produce CryI proteins (active against lepidopterous insects) and another gene that produces a CryIII protein (active against coleopterous insects). This strategy is one of genetic recombination, but it does not involve recombinant DNA technology.

Mycogen Corporation, in San Diego, California, has used recombinant DNA technology to produce strains of the bacterium *Pseudomonas fluorescens* that produce modified Cry proteins (Gelernter and Schwab 1993). Producing the proteins in *P. fluorescens* provides some protection from UV degradation under field conditions. As part of the manufacturing process, the *P. fluorescens* cells are killed before being formulated into an insecticidal powder or liquid (the product). Recombinant DNA technology has also been used to produce a wide variety of insecticidal transgenic crop plants that produce CryI and CryIII proteins (Fischhoff et al. 1989, Perlak et al. 1990). Among the viruses, AcNPV has been genetically engineered to produce insect-selective neurotoxins and enzymes, for example, juvenile hormone esterase (Hawtin and Possee 1993). The goal is to improve the efficacy of the NPVs by enabling them to paralyze caterpillar hosts quickly to prevent feeding damage. The apparent host range of these faster acting recombinant NPVs might be slightly expanded to include additional lepidopteran species that do not normally succumb to viral infection. In these less susceptible species, although the virus should not replicate any better, the production of potent neurotoxins could kill a greater range of species than that killed by the wild-type virus. Recombinant viruses have not yet been registered by EPA for commercial use. The evaluation and use of recombinant insect-pathogenic fungi and microsporidia are also years away.

Most questions about the host specificity of genetically modified agents can be addressed with empirical laboratory testing. EPA is responsible for designing and evaluating such tests. For *Bt* and viruses, the objective of these tests is to ensure that the host range has not been extended beyond that intended. Thus, a wide range of target and nontarget insect and noninsect invertebrates as well as vertebrates are tested for their sensitivity and susceptibility to genetically modified or engineered pathogens. With the *Bt* strains tested to date, no unanticipated expansion of the host range has been detected. With NPVs, only preliminary testing has been done. However, these tests indicate no significant increase in host range; whereas additional species within the insect order *Lepidoptera* may be susceptible to a modified virus but not its wild-type parent, expansion of the host range to insects of other orders is unlikely.

Empirical laboratory tests are not designed to evaluate the consequences of long-term use. In the case of *Bt*, consequences are likely to be minor, if any, because the genetically modified and recombinant

strains should be as labile as those from which they were derived. In the case of viruses, NPVs are being designed to deteriorate rapidly in the environment, a tactic that recent studies indicate should be effective (Wood et al. 1994). For the fungi and microsporidia, genetic engineering studies have only recently been initiated, and it is too early to predict potential consequences because the specific traits to be modified have not been determined.

#### LABORATORY HOST RANGE VERSUS ECOLOGICAL HOST RANGE

Most information about host specificity is based on laboratory experiments, yet concerns are almost always about host specificity in the environment (i.e., ecological host range). This situation is true for all biological control organisms, especially nonindigenous organisms introduced as classical biological control agents. It is generally accepted that the host range determined in the laboratory will be much broader than ecological host range, because in laboratory tests nontarget organisms are exposed to much higher concentrations of the test pathogen than they would normally encounter in nature. However, laboratory tests lack the realism of field experiments because they often fail to capture the genetic variability present in the total population of a candidate pathogen or nontarget organism.

In making decisions about how, when, and where to use pathogens for insect control, the phylogenetic distribution of diseases among insect and noninsect species becomes relevant because it represents the interactions of host and pathogen populations over millions of years. Thus, it can serve as an indicator of the types of shifts or expansions in host range that could occur from increased use of a pathogen or from the introduction of nonindigenous pathogens. Nevertheless, it would be useful to develop data sets to compare the results of laboratory host specificity studies with the host specificity of the same pathogen in the field over time.

#### CONCLUSIONS

The use of bacteria to control insects, especially various subspecies of Bt, is likely to continue to grow, without any major shifts or expansions in host range other than those made deliberately through genetic engineering. Bacteria will continue to be used as microbial insecticides (Bt) or augmentative releases (*B. popilliae*). Cry proteins from Bt are also likely to be used in transgenic insecticidal plants. This use may lead to changes in the efficacy of specific proteins against specific pests, but there is no reason to think it will result in shifts or expansions of host range because the opportunities for Cry gene recombination are minimal and restricted to crop plants.

The use of NPVs as microbial insecticides against lepidopterous insect pests in which Bt products are ineffective should increase. Shifts or expansions in host range are unlikely in this case as well, but they cannot be completely ruled out for those NPVs that already have a broad host range and may be used repeatedly in the same habitat (Fuxa 1990, Wood et al. 1994). Genetically engineered viruses that are more virulent but more environmentally labile should eliminate any potential for host range shifts or expansions.

The probability of host range expansion among fungal entomopathogens is difficult to evaluate or predict. Because fungi have not been applied extensively as microbial insecticides, we have little information on the effects on nontarget hosts of continued exposure to inundative applications of a specific fungal biotype. Most fungi are likely to infect more nontarget hosts under optimal conditions in the laboratory than under field conditions. Inundative field applications of a fungus should theoretically have a higher probability of causing infections in nontarget host species than the same fungal biotype occurring naturally in the field, because large amounts of conidia will be directly applied to many insect species that do not normally encounter such loads. If horizontal transmission occurred within a nontarget host species infected as a result of inundative applications, selection pressure on the fungus could allow it permanently

to colonize the nontarget host species. These possibilities are all speculative. To intelligently address these issues, we need more information on the identity and ecology of fungal biotypes that cause disease outbreaks and on the population genetics of the fungal biotypes.

Questions involving host range expansion of microsporidian species are similar to the questions posed for fungal pathogens. Microsporidia have seldom been used as microbial insecticides, and there is no information about how nontarget hosts will be affected by continued exposure to inundative applications of microsporidia. More information is needed on the taxonomy, biotypes, ecology, and population genetics of microsporidia that occur naturally in field populations of insects to evaluate host range expansion of microsporidia.

Although we have little information on the effects of inundative applications of viral, fungal, and microsporidian pathogens of insects, we do have extensive data sets on the occurrence of diseases in many species of pest insects (Campbell and Podgwaite 1971, Maddox 1987, Siegel et al. 1988). These data strongly suggest that, under field conditions, insect pathogens (including nonindigenous species) do not move readily from one host to another, even in sympatric host species. Nevertheless, host range expansion is a part of the natural evolutionary process, and the rate at which it occurs is undoubtedly influenced by many variables. But over the short term, there is no evidence to suggest that the additional use of pathogens to control insects will lead to significant shifts in host range and concomitant detrimental consequences. The challenge is to avoid creating a situation in which host range expansion or host switching becomes an environmental problem. The best way to achieve this goal is to develop a sound understanding of host specificity and how the tactics we use for biological control influence the host specificity of biocontrol agents. Nevertheless, there is no evidence that suggests we should substantially restrict the use of pathogens to control insect pests. To paraphrase a remark made by Carlo M. Ignoffo (1975) more than 20 years ago with respect to the use of NPVs in pest control: We must be extremely careful that our prudence does not backfire--that it does not become too scrupulous and result in failure to act. Such an attitude could seriously jeopardize one of our most attractive alternatives to chemical pesticides.

#### Added material

Brian A. Federici is a professor in the Department of Entomology, and a member of the Interdepartmental Graduate Program in Genetics, University of California, Riverside, CA 92521. Joseph V. Maddox is an entomologist in the Center for Economic Entomology, Illinois Natural History Survey, Champaign, IL 61820. © 1996 American Institute of Biological Sciences.

Table 1. Specific activity of *Bacillus thuringiensis* CryIA proteins against certain lepidopterous species. Data from Hofte and Whiteley (1989). LC50 is the concentration required to cause 50[percent] mortality in a **treated** larval population; the lower the value, the more potent the protein. The concentrations indicated here are in terms of nanograms of protein per square centimeter of artificial diet surface. The common names of the insects are as follows: *Manduca sexta*, tomato hornworm; *Heliothis virescens*, cotton bollworm; *Mamestra brassicae*, European cabbage worm.

Table 2. Examples of commercially available microbial insecticides based on *Bacillus thuringiensis*.

Figure 1. Parasporal bodies of typical subspecies of *Bacillus thuringiensis* in commercial formulations of this insecticidal bacterium. (a) Phase contrast light micrograph of sporulating cells of *B. thuringiensis*. The arrows point to the parasporal body, the structure that contains insecticidal proteins. (b) Scanning electron micrograph of the parasporal bodies from the HD1 isolate of *B. thuringiensis* subsp. *kurstaki*. Each bipyramidal crystal contains three CryIA proteins (a, b, and c); each cuboidal crystal contains a single protein, CryIIA. (c) Transmission electron micrograph of the parasporal body of *B. thuringiensis* subsp. *israelensis*. The parasporal body of this subspecies, which is toxic to mosquito and blackfly larvae, contains four proteins, CryIVA, B, D, and CytA. Parasporal bodies contain inclusions of different electron densities, each composed of a different insecticidal protein. The

arrowheads point to the fibrous matrix that holds the inclusions together.

Figure 2. Light and electron micrographs of nuclear polyhedrosis viruses, the most common virus type used for insect control. (a) Fat body cells of an infected caterpillar. The arrows point to cells at different stages of disease progression, from early (left) to advanced (right). At the termination of cellular disease, each nucleus is filled with virion-containing polyhedra (red granules). (b) Tracheal matrix cells of an infected caterpillar illustrating clusters of polyhedra in infected nuclei adjacent to trachea. (c) Transmission electron micrograph illustrating the structure and distribution of virions in a single polyhedron. Billions of polyhedra are produced by the virus in each insect larva. The virions are the dense rod-shaped structures surrounded by a protein matrix. The latter dissolve in the insect stomach, releasing the virions, which then infect the insect through the midgut (stomach) lining.

Figure 3. Fungal conidia of an entomophthoraceous fungus and a caterpillar showing the typical signs of mycosis after death. (a) The conidia typically attach to the insect cuticle and penetrate the body wall via a germ tube. After colonizing the body, reproductive hyphae pass out through the body wall and produce conidia on the surface of the dead insect. (b) The white bands on this dead caterpillar are clusters of conidiophores, which produce conidia at their tips.

Figure 4. Light (a) and electron (b) micrographs illustrating the structure of typical microsporidian spores. Ingested spores infect the insect by **injecting** the sporoplasm through the midgut wall and into the insect body via the polar filament, a tubular structure coiled like a bedspring within the spore. The arrowheads and arrows point to sections through the polar filament at different positions in the spore. The clear central area is the nucleus.

#### REFERENCES CITED

- Balch RE, Bird FT. 1944. A disease of the European spruce sawfly, *Gilpinia hercyniae* [Htg.] and its place in natural control. *Scientific Agriculture* 25: 65-80.
- Betz FS, Forsyth SF, Stewart WE. 1990. Registration requirements and safety considerations for microbial pest control agents. Pages 3-10 in Laird M, Lacey LA, Davidson EW, eds. *Safety of microbial insecticides*. Boca Raton (FL): CRC Press.
- Boucias DG, Latge JP. 1988. Nonspecific induction of germination of *Conidiobolus obscurus* and *Nomuraea rileyi* with host and non-host cuticle extracts. *Journal of Invertebrate Pathology* 43: 288-292.
- Boucias DG, Pendland JC. 1984. Nutritional requirements for conidial germination of several host range pathotypes of the entomopathogenic fungus *Nomuraea rileyi*. *Journal of Invertebrate Pathology* 43: 288-292.
- Boucias DG, Pendland JC, Latge JP. 1988. Nonspecific factors involved in attachment of entomopathogenic Deuteromycetes to host insect cuticle. *Applied and Environmental Microbiology* 54: 1795-1805.
- Brooks WM. 1988. Entomogenous protozoa. Pages 1-149 in Ignoffo CM, Mandava NB, eds. *Handbook of natural pesticides*. Vol. 5. *Microbial pesticides*. Part A: Entomogenous protozoa and fungi. Boca Raton (FL): CRC Press.
- Campbell RW, Podgwaite JD. 1971. The disease complex of the gypsy moth. Major components. *Journal of Invertebrate Pathology* 18: 101-107.
- Cameron JWM. 1973. Insect pathology. Pages 285-306 in Smith RF, Mittler TH, Smith CN, eds. *History of entomology*. Palo Alto (CA): Annual Reviews.
- Canning EU. 1982. An evaluation of protozoal characteristics in relation to biological control of pests. *Parasitology* 84: 119-129.
- Canning EU. 1990. Phylum Microspora. Pages 53-72 in Margulis L, Corliss JO, Melkonian M, Chapman DJ, eds. *Handbook of Protista*. Boston (MA): Jones and Bartlett.
- Croizier G, Croizier L, Arguard O, Poudevigne D. 1994. Extension of *Autographa californica* nuclear polyhedrosis virus host range by interspecific replacement of a short DNA sequence in the p 143 helicase gene. *Proceedings of the National Academy of Sciences of the United States*

of America 91: 48-52.

de Barjac H, Frachon E. 1990. Classification of *Bacillus thuringiensis* strains. *Entomophaga* 35: 233-240.

Fargues J. 1976. Specificite des champignons pathogenes imparfaits (Hyphomycetes) pour les larves de Coleopteres (Scarabaeidae et Chrysomelidae). *Entomophaga* 21: 313-323.

Fargues J, Remaudiere G. 1977. Considerations on the specificity of entomopathogenic fungi. *Mycopathologia* 62: 31-40.

Federici BA. 1993. Viral pathobiology in relation to insect control. Pages 81-101 in Beckage NE, Thompson SN, Federici BA, eds. *Parasites and pathogens of insects*. Vol. 2. San Diego (CA): Academic Press.

Ferron P. 1981. Pest control by the fungi *Beauveria* and *Metarhizium*. Pages 465-482 in Burges HD, ed. *Microbial control of pests and plant diseases*. London (UK): Academic Press.

Ferron P, Hurpin B, Robert PH. 1972. Sur la specificite de *Metarhizium anisopliae* (Metsch.) Sorokin. *Entomophaga* 17: 165-178.

Fischhoff DA, et al. 1987. Insect tolerant transgenic tomato plants. *Bio/Technology* 5: 807-813.

Fuxa JR. 1990. Environmental risks of genetically engineered entomopathogens. Pages 203-207 in Laird M, Lacey LA, Davidson EW, eds. *Safety of microbial insecticides*. Boca Raton (FL): CRC Press.

Ge AZ, Shivarova NI, Dean DH. 1989. Location of the *Bombyx mori* specificity domain on a *Bacillus thuringiensis* -endotoxin protein. *Proceedings of the National Academy of Sciences of the United States of America* 86: 4037-4041.

Gelernter WD, Schwab GE. 1993. Transgenic bacteria, viruses, algae, and other microorganisms as *Bacillus thuringiensis* toxin delivery systems. Pages 89-104 in Entwistle PF, Cory JS, Bailey MJ, Higgs S, eds. *Bacillus thuringiensis, an environmental biopesticide: theory and practice*. Chichester (UK): John Wiley & Sons.

Goettel MS, Poprawski JD, Vanderberg JD, Li Z, Roberts DW. 1990. Safety to nontarget invertebrates of fungal biocontrol agents. Pages 209-231 in Laird M, Lacey LA, Davidson EW, eds. *Safety of microbial insecticides*. Boca Raton (FL): CRC Press.

Groner A. 1986. Specificity and safety of baculoviruses. Pages 177-202 in Granados RR, Federici BA, eds. *The biology of baculoviruses*. Vol. I: biological properties and molecular biology. Boca Raton (FL): CRC Press.

Groner A. 1990. Safety to nontarget invertebrates of baculoviruses. Pages 135-147 in Laird M, Lacey LA, Davidson EW, eds. *Safety of microbial insecticides*. Boca Raton (FL): CRC Press.

Harcourt D, Guppy GMJC, Macleod DM, Tyrell D. 1974. The fungus *Entomophthora phytonomi* pathogenic to the alfalfa weevil, *Hypera postica* (Coleoptera: Curculionidae). *Canadian Entomologist* 109: 1521-1532.

Hawtin RE, Possee RD. 1993. Genetic manipulation of the baculovirus genome for pest control. Pages 179-195 in Beckage NE, Thompson SN, Federici BA, eds. *Parasites and pathogens of insects*. Vol. 2. San Diego (CA): Academic Press.

Henry JE, Onsager JA. 1982. Large-scale test of control of grasshoppers on rangeland with *Nosema locustae*. *Journal of Economic Entomology* 75: 31-35.

Hofte H, Whiteley HR. 1989. Insecticidal crystal proteins of *Bacillus thuringiensis*. *Microbiology Reviews* 53: 242-255.

Ignoffo CM. 1975. Evaluation of in vivo specificity of insect viruses, 1975. Pages 52-57 in Summers MD, Engler R, Falcon L, Vail P, eds. *Baculoviruses for insect pest control: safety considerations*. Washington (DC): American Society for Microbiology.

Klein MG. 1981. Advances in the use of *Bacillus popilliae* for pest control. Pages 183-192 in Burges HD, ed. *Microbial control of pests and plant diseases 1970-1980*. New York: Academic Press.

Lacey LA, Mulla MS. 1990. Safety of *Bacillus thuringiensis* spp. *israelensis* and *Bacillus sphaericus* to nontarget organisms in the aquatic environment. Pages 169-188 in Laird M, Lacey LA, Davidson EW, eds. *Safety of microbial insecticides*. Boca Raton (FL): CRC Press.

Li J, Carroll J, Ellar DJ. 1991. Crystal structure of insecticidal



-endotoxin from *Bacillus thuringiensis* at 2.5 angstrom resolution. *Nature* 353: 815-821.

Maeda S, Kamita SG, Kondo A. 1993. Host range expansion of *Autographa californica* nuclear polyhedrosis virus (NPV) following recombination of a 0.6-kilobase-pair DNA fragment originating from *Bombyx mori*. *Journal of Virology* 67: 6234-6238.

Maddox JV. 1987. Protozoan diseases. Pages 417-452 in Fuxa JR, Tanada Y, eds. *Epizootiology of insect diseases*. New York: John Wiley.

Maddox JV, Brooks WM, Fuxa JR. 1981. *Vairimorpha necatrix*, a pathogen of agricultural pests: potential for pest control. Pages 587-594 in Burges HD, ed. *Microbial control of pests and plant diseases*. London (UK): Academic Press.

Martignoni ME, Iwai PJ. 1986a. A catalog of viral diseases of insects, mites, and ticks. 4th ed. General Technical Reports nr PNW-195. Washington (DC): US Forest Service.

Martignoni ME, Iwai PJ. 1986b. Propagation of multinucleocapsid nuclear polyhedrosis virus of *Orygia pseudotsugata* in larvae of *Trichoplusia ni*. *Journal of Invertebrate Pathology* 47: 32-41.

McCoy CW, Samson RA, Boucias DG. 1988. Entomogenous fungi. Pages 151-236 in Ignoffo CM, Mandava NB, eds. *Handbook of natural pesticides*. Vol. V. Microbial pesticides. Part A: entomogenous protozoa and fungi. Boca Raton (FL): CRC Press.

Meadows MP. 1993. *Bacillus thuringiensis* in the environment: ecology and risk assessment. Pages 193-220 in Entwistle PF, Cory JS, Bailey MJ, Higgs S, eds. *Bacillus thuringiensis*, an environmental biopesticide: theory and practice. Chichester (UK): John Wiley & Sons.

Miller JC. 1990. Field assessment of the effects of a microbial pest control agent on nontarget lepidoptera. *American Entomologist* 36: 135-139.

Perlak FJ, Deaton TA, Armstrong RL, Fuchs SR, Sims JT, Greenplate JT, Fischhoff DA. 1990. Insect resistant cotton plants. *Bio/Technology* 8: 939-943.

Pietrantonio PV, Federici BA, Gill SS. 1993. Interaction of *Bacillus thuringiensis* endotoxins with the insect midgut epithelium. Pages 55-79 in Beckage NE, Thompson SN, Federici BA, eds. *Parasites and pathogens of insects*. Vol. 2. San Diego (CA): Academic Press.

Ramoska WA, Todd T. 1985. Variation in efficacy and viability of *Beauveria bassiana* in the chinch bug as the result of feeding activity on selected host plants. *Environmental Entomology* 14: 146-150.

Samson RA, Evans HC, Latge JP. 1988. *Atlas of entomopathogenic fungi*. Berlin (Germany): Springer-Verlag.

Sangadala S, Walters FS, English LH, Adang MJ. 1994. A mixture of *Manduca sexta* aminopeptidase and phosphatase enhances *Bacillus thuringiensis* insecticidal CryIA(c) toxin binding and 86Rb(+)-K<sup>+</sup> efflux in vitro. *Journal of Biological Chemistry* 269: 10088-10092.

Savage KE. 1975. *Nosema algerae* Vavra and Undeen, 1970 (Protozoa: Microsporidia): its bionomics and development for use as a biological control agent of mosquitoes. [MS thesis.] University of Florida, Gainesville, FL.

Siegel JP, Maddox JV, Ruesink WG. 1988. Seasonal progress of *Nosema pyrausta* in the European corn borer, *Ostrinia nubilalis*. *Journal of Invertebrate Pathology* 52: 130-136.

Steffey KL, Armbrust EJ, Onstad DW. 1994. Management of insects in alfalfa. Pages 469-506 in Metcalf RL, Luckmann WH, eds. *Introduction to insect pest management*. New York: John Wiley and Sons.

St. Leger R. 1993. Biology and mechanisms of insect-cuticle invasion by Deuteromycete fungal pathogens. Pages 211-229 in Beckage NE, Thompson SN, Federici BA, eds. *Parasites and pathogens of insects*. Vol. 2. San Diego (CA): Academic Press.

Tanada Y, Fuxa JR. 1987. The pathogen population. Pages 113-157 in Fuxa JR, Tanada Y, eds. *Epizootiology of insect diseases*. New York: John Wiley & Sons.

Thomas ED, Heimpel AM, Adams JR. 1974. Determination of the active nuclear polyhedrosis virus content of untreated cabbages. *Environmental Entomology* 3: 908-910.

Undeen AH, Maddox JV. 1973. The infection of nonmosquito hosts by

**injection** with spores of the microsporidian *Nosema algerae*. Journal of Invertebrate Pathology 22: 258-265.

Wood HA, Hughes PR, Shelton A. 1994. Field studies of the co-occlusion strategy with a genetically altered isolate of the *Autographa californica* nuclear polyhedrosis virus. Environmental Entomology 23: 211-219.

10/7/16 (Item 1 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

10796138 20324401 PMID: 10868549

Anticoccidial **vaccination** of broiler chickens in various management programmes: relationship between oocyst accumulation in litter and the development of protective immunity.

Williams R B; Johnson J D; Andrews S J  
Schering-Plough Animal Health, Harefield, Uxbridge, UK.  
ray.williams@spcorp.com

Veterinary research communications (NETHERLANDS) Jul 2000, 24 (5)  
p309-25, ISSN 0165-7380 Journal Code: 8100520

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Paracox anticoccidial **vaccine** was administered to a 7-day-old flock of commercial broiler breeder stock subsequently reared to point-of-lay in the same house. For comparison, three subgroups of another flock of broiler breeders were also **vaccinated** with Paracox at 7 days of age, reared to 42 days and then transferred to new litter on another farm until point-of-lay. The first subgroup received no further **treatment**, but the second and third each received a second **vaccination** with Paracox, either immediately after transfer to the new litter or 42 days after transfer. Using an *Eimeria necatrix* model, protective immunity was demonstrated by virulent challenge of samples of birds from all groups by the age of 37-40 days (30-33 days after the first **vaccination**), and was maintained to at least 122-125 days of age, whether the birds remained on the same litter or were transferred to another farm, and whether they received one or two anticoccidial **vaccinations**. Therefore, there is no disadvantage in transferring birds onto new litter 35 days after a single Paracox **vaccination**, nor is there any advantage in giving a second **vaccination** after such a transfer. **Vaccinated** birds seeded the new litter with oocysts, despite being clinically immune to coccidiosis. A supplementary laboratory experiment showed that birds **vaccinated** at 8 days of age passed almost no oocysts after a second **vaccination** at 43 days of age. This indicated that they were not only protected against clinical coccidiosis, but were almost solidly immune to a homologous infection 5 weeks after a single **vaccination**. Nevertheless, oocysts appeared in the litter of all four groups of commercial breeders throughout the trial, showing that wild-type heterologous infections occurred whether the birds were transferred to new litter or not, but these did not overwhelm the acquired protective immunity and cause clinical coccidiosis.

Record Date Created: 20001019

10/7/17 (Item 2 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

02089060 75161155 PMID: 165646

Pathogenic mechanisms not operating in *Eimeria necatrix* infections.

Ryley J F

Zeitschrift fur Parasitenkunde (Berlin, Germany) (GERMANY, WEST) 1975,  
45 (3) p269-79, ISSN 0044-3255 Journal Code: 8710749

Document type: Journal Article

Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed

Having investigated certain aspects of **Eimeria necatrix** coccidiosis in chickens, workers of the Hannover Veterinary School postulated "that death following a single inoculation of a large number of oocysts is due to an alarm reaction and not a specific pathogenic action of the parasites". Because this hypothesis is somewhat revolutionary in its concept, several pieces of evidence on which it is based and several logical deductions which can be made from it have been examined. It has been confirmed that **injection** of chickens with cysteamine or 5-hydroxytryptamine 30 min before inoculation of the birds with a lethal dose of **E. necatrix** oocysts reduces subsequent mortality; the reason for this, however, appears not to be the neutralisation of the proposed shock reaction, but rather an inhibition of the excystation process, brought about indirectly through the host. Inoculation of chickens with a non-lethal dose of **E. necatrix** oocysts 30 min before inoculation with a lethal dose of oocysts was followed by increased mortality rather than the decreased mortality which the hypothesis would predict. **Treatment** of chickens with sulphadimidine starting 48 h after inoculation resulted in survival of the birds rather than death which would ensue if in fact mortality was due to a shock reaction irreversibly initiated at the time of inoculation. A direct effect of sulphadimidine on the parasite has been shown both in vivo and in vitro.

Record Date Created: 19750731

10/7/18 (Item 1 from file: 370)  
DIALOG(R)File 370:Science  
(c) 1999 AAAS. All rts. reserv.

00508131 (USE 9 FOR FULLTEXT)  
Experimental Evolution of Parasites  
Ebert, Dieter  
The author is at the Universitaet Basel, Zoologisches Institut, Rheinsprung 9, 4051 Basel, Switzerland. E-mail: ebert@ubaclu.unibas.ch  
Science Vol. 282 5393 pp. 1432  
Publication Date: 11-20-1998 (981120) Publication Year: 1998  
Document Type: Journal ISSN: 0036-8075  
Language: English  
Section Heading: comcopy  
Word Count: 3062

**Abstract:** Serial passage experiments are a form of experimental evolution that is frequently used in applied sciences; for example, in **vaccine** development. During these experiments, molecular and phenotypic evolution can be monitored in real time, providing insights into the causes and consequences of parasite evolution. Within-host competition generally drives an increase in a parasite's virulence in a new host, whereas the parasite becomes avirulent to its former host, indicating a trade-off between parasite fitnesses on different hosts. Understanding why parasite virulence seldom escalates similarly in natural populations could help us to manage virulence and deal with emerging diseases

**References and Notes:**

1. Williams, G. C., Nesse, R. M., Q. Rev. Biol., 66 1991, 1 ;
2. Bull, J. J., Evolution, 48 1994, 1423 ;
3. Ewald, P. W., The Evolution of Infectious Disease, 1994, Oxford Univ. Press, Oxford, ;
4. Woolcock, P. R., Crighton, G. W., Vet. Rec., 105 1979, 30 ;
5. Brugh, M., Perdue, M. L., Avian Dis., 35 1991, 824 ;
6. Jinks, J. L., Grindley, M., Heredity, 18 1963, 245 ;
7. Gutekunst, D. E., Becvar, C. S., Am. J. Vet. Res., 40 1979, 974 ;
8. Dobson, C., Owen, M. E., Int. J. Parasitol., 7 1977, 463 ;
9. Cunfer, B. M., Ann. Appl. Biol., 104 1984, 6110. Sutherland, I.

- A., et.al. Exp. Parasitol., 83 1996, 125 ;
11. Wong, M. M., Karr, S. L., Chow, C. K., J. Parasitol., 63 1977, 872 ;
12. Berge, T. O., Banks, I. S., Tigertt, W. D., Am. J. Hyg., 73 1961, 209 ;
13. Callow, L. L., Mellors, L. T., McGregor, W., Int. J. Parasitol., 9 1979, 333 ;
14. Bawden, F. C., J. Gen. Microbiol., 18 1958, 751 ;
15. Pavan, O. H., Boucias, D. G., Pendland, J. C., Entomophaga, 26 1981, 99 ;
16. Carson, C. A., Timms, P., Cowman, A. F., Stewart, N. P., Exp. Parasitol., 70 1990, 404 ;
17. Marchette, N. J., et.al. Am. J. Trop. Med. Hyg., 43 1990, 212 ;
18. Muskett, J. C., Reed, N. E., Thornton, D. H., **Vaccine**, 3 1985, 30919. Mansky, L. M., J. Gen. Virol., 79 1998, 1337 ;
20. Diez, J., Hofner, M., Domingo, E., Donaldson, A. I., Virus Res., 18 1990, 3 ;
21. Novella, I. S., et.al. Proc. Natl. Acad. Sci. U.S.A., 92 1995, 5841 ;
22. LeClerc, J. E., Li, B., Payne, W. L., Cebula, T. A., Science, 274 1996, 1208 ;
23. Mel, S. F., Mekalanos, J. J., Cell, 87 1996, 795 ;
24. Bull, J. J., Molineux, I. J., Evolution, 46 1992, 882 ;
25. Ni, Y., Kemp, M. C., J. Gen. Virol., 73 1992, 3107 ;
26. Kumar, S., Miller, L. K., Virus Res., 7 1987, 335 ;
27. Olmsted, R. A., Baric, R. S., Sawyer, B. A., Johnston, R. E., Science, 225 1984, 424 ;
28. Kurath, G., Dodds, J. A., RNA, 1 1995, 491 ;
29. Proutski, V., Gaunt, M. W., Gould, E. A., Holmes, E. C., J. Gen. Virol., 78 1997, 1543 ;
30. Westrop, G. D., et.al. J. Virol., 63 1989, 1338 ;
31. Takai, S., et.al. Vet. Microbiol., 39 1994, 187 ;
32. Groisman, E. A., Ochman, H., Cell, 87 1996, 791 ;
33. Sabin, A., Hennessen, W. A., Winsor, J., J. Exp. Med., 99 1954, 551 ;
34. Chao, L., Nature, 348 1990, 454 Clarke, D. K., et.al. J. Virol., 67 1993, 222 ;
35. Baumann, P., Moran, N., Baumann, L., BioScience, 47 1997, 12 Moran, N., Proc. Natl. Acad. Sci. U.S.A., 93 1996, 2873 ;
36. Brown, A. J. Leigh, Proc. Natl. Acad. Sci. U.S.A., 94 1997, 1862 ;
37. Herre, E. A., Parasitology, 111 1995, S179 ;
38. Nowak, M. A., May, R. M., Proc. R. Soc. London Ser. B, 255 1994, 81 ;
39. Diffley, P., Scott, J. O., Mama, K., Tsen, T. N. R., Am. J. Trop. Med. Hyg., 36 1987, 533 ;
40. Ebert, D., Mangin, K. L., Evolution, 51 1997, 1828 ;
41. Lipsitch, M., Moxon, E. R., Trends Microbiol., 5 1997, 31 ;
42. Turner, P. E., Cooper, S. C., Lenski, R. E., Evolution, 52 1998, 315 ;
43. Dearsly, A. L., Sinden, R. E., Self, I. A., Parasitology, 100 1990, 359 ;
44. Donges, J., Parasitologie, 1988, Thieme, Stuttgart, Germany, ;
45. Contreras, V. T., Araque, W., Delgado, V. S., Mem. Inst. Oswaldo Cruz, 89 1994, 253 ;
46. Fry, J. D., Am. Nat., 136 1990, 569 Gould, F., Evolution, 33 1979, 791 ;
47. Johnston, R. E., Smith, J. F., Virology, 162 1988, 437 ;
48. Zelle, M. R., J. Infect. Dis., 71 1942, 131 ;
49. Ebert, D., Ed. by Stearns, S. C., Evolution in Health and Disease, 1999, 161172 Oxford Univ. Press, Oxford, ;
50. Anderson, R. M., May, R. M., Parasitology, 85 1982, 411 ;
51. Ebert, D., Hamilton, W. D., Trends Ecol. Evol., 11 1996, 79 ;
52. Adams, M. W., Ellinghoe, A. H., Rossmann, E. C., Bioscience, 21

- 1971, 1067 ;
53. Jaenike, J., *Evol. Theory*, 3 1978, 191 Hamilton, W. D., *Oikos*, 35 1980, 282 Lively, C. M., *Bioscience*, 46 1996, 107 ;
  54. Stevens, N. E., *J. Am. Soc. Agron.*, 40 1948, 841 Burdon, J. J., *J. Appl. Ecol.*, 18 1981, 649 ;
  55. Liersch, S., Schmid-Hempel, P., *Proc. R. Soc. London Ser. B*, 265 1998, 1 ;
  56. Day, K. P., et.al. *Proc. Natl. Acad. Sci. U.S.A.*, 90 1993, 8292 ;
  57. Harrower, K. M., *Trans. Br. Mycol. Soc.*, 68 1977, 101 ;
  58. Smith, J. M., *Am. Nat.*, 100 1966, 637 Felsenstein, J., *Evolution*, 35 1981, 124 ;
  59. Via, S., *Annu. Rev. Entomol.*, 35 1990, 421 Mackenzie, A., *Evolution*, 50 1996, 155 ;
  60. Bull, J. J., Molineux, I. J., Rice, W. R., *Evolution*, 45 1991, 875 ;
  61. Kilbourne, E. D., Murphy, J. S., *J. Exp. Med.*, 111 1960, 337 ;
  62. Janda, Z., Vonka, V., *Arch. Gesamte Virusforsch.*, 24 1968, 192 ;
  63. Theiler, M., Smith, H. H., *J. Exp. Med.*, 65 1937, 782 ;
  64. Rubin, S. A., Waltrip, R. W., Bautista, J. R., Carbone, K. M., *J. Virol.*, 67 1993, 548 ;
  65. Karron, R. A., et.al. *J. Infect. Dis.*, 157 1988, 338 ;
  66. Cao, J. X., et.al. *J. Gen. Virol.*, 76 1995, 2757 ;
  67. Plowright, W., *Rev. Sci. Tech. Off. Int. Epizoo.*, 5 1986, 897 ;
  68. Zuckerman, M. A., Rebecca, J. C., Oxford, J. S., *J. Infect.*, 28 1994, 41 ;
  69. Levine, J. F., et.al. *Res. Vet. Sci.*, 48 1990, 64 ;
  70. Turnbull, P. C. B., **Vaccine**, 9 1991, 533 ;
  71. Catrenich, E. E., Johnson, W., *Infect. Immun.*, 56 1988, 3121 ;
  72. Brummer, E., Restrepo, A., Hanson, L. H., Stevens, D. A., *Mycopathologia*, 109 1990, 13 ;
  73. Levin, H. B., Pappagianis, D., Cobb, J. M., *Mycopathol. Mycol. Appl.*, 41 1979, 177 ;
  74. Kogut, M. H., Gore, T. C., Long, P. L., *Parasitology*, 86 1983, 199 ;
  75. Chin, W., Contacos, P. G., Collins, W. E., Jeter, M. H., Alpert, E., *Am. J. Trop. Med. Hyg.*, 17 1968, 355 ;
  76. Lecompte, V., Chumpitazi, B. F. F., Pasquier, B., Ambroise-Thomas, P., Santoro, F., *Parasitol. Res.*, 78 1992, 26777. Guthrie, W. D., Rathore, Y. S., Cox, D. F., Reed, G. L., *J. Econ. Entomol.*, 67 1974, 605 ;
  78. Claridge, M. F., Hollander, J., *Entomol. Exp. Appl.*, 32 1982, 213 ;
  79. I thank A. Carius, J. Hottinger, T. Kaweck, R. Lenski, T. Little, P. Schmid-Hempel, S.C. Stearns, and N. Sokolova for help and encouragement during various steps in the preparation of this paper. I am supported by the Swiss Nationalfond (grant no. 3100-043093.95).

10/7/19 (Item 1 from file: 399)  
 DIALOG(R)File 399:CA SEARCH(R)  
 (c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

112005940 CA: 112(1)5940x PATENT  
 Method of treating a bird's egg with an immunogen for immunization and eggs treated thereby  
 INVENTOR(AUTHOR): Smith, Harold V.; Thaxton, J. Paul; Ruff, Michael D.  
 LOCATION: USA  
 ASSIGNEE: Embrex, Inc.; United States Dept. of Agriculture  
 PATENT: European Pat. Appl. ; EP 291173 A2 DATE: 881117  
 APPLICATION: EP 88303349 (880414) \*US 39052 (870416)  
 PAGES: 9 pp. CODEN: EPXXDW LANGUAGE: English CLASS: A61K-039/012A  
 DESIGNATED COUNTRIES: AT; BE; CH; DE; ES; FR; GB; GR; IT; LI; LU; NL; SE  
 SECTION:

CA215002 Immunochemistry

IDENTIFIERS: bird immunization antigen embryo, chicken immunization  
Eimeria egg, protein Eimeria immunization chicken egg, coccidiosis  
immunization chicken egg

DESCRIPTORS:

Eimeria tenella... Eimeria...

antigens of, immunization of chick embryo with

Antigens...

embryo of bird immunization with

Coccidiosis...

embryo of chicken immunization to, antigens for

Duck... Goose... Pheasant... Quail... Turkey...

immunization of embryo of, antigens for

Bird... Chicken...

immunization of embryo of, to coccidiosis and other diseases

Embryo...

immunization of, of bird, antigens for

Immunization...

of embryo, of bird, antigens for

Antibodies...

to Eimeria tenella, detn. of, by ELISA

CAS REGISTRY NUMBERS:

124204-12-2D fusion products with .beta.-galactosidase, embryo of chicken  
immunization with

9031-11-2D fusion products with Eimeria tenella antigen, embryo of chicken  
immunization with

? ds

Set	Items	Description
S1	1547	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S5	17	S4 NOT S3
S6	36420	(EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)
S7	984	S6 AND (OVO OR EGG?)
S8	120	S7 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S9	72	RD S8 (unique items)
S10	19	(TREAT? OR DISRUPT? OR GRIND?) AND S9
? s s9 not (s3 or s5 or s10)		
	72	S9
	14	S3
	17	S5
	19	S10
S11	53	S9 NOT (S3 OR S5 OR S10)

? t s11/7/all

>>>Format 7 is not valid in file 143

11/7/1 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

11252391 BIOSIS NO.: 199800033723

Live attenuated **vaccines** against avian coccidiosis: Success with  
precocious and **egg**-adapted lines of **Eimeria**.

AUTHOR: Shirley M W(a); Bedrnik P

AUTHOR ADDRESS: (a)Inst. Anim. Health, Compton Lab., Compton, Nr Newbury,  
Berkshire RG20 7NN\*\*UK

JOURNAL: Parasitology Today 13 (12):p481-484 Dec., 1997

ISSN: 0169-4758

DOCUMENT TYPE: Article

RECORD TYPE: Citation

LANGUAGE: English

11/7/2 (Item 2 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

11252378 BIOSIS NO.: 199800033710  
The importance of transmission-blocking immunity in the control of  
infections by apicomplexan parasites.  
AUTHOR: Wallach Michael(a)  
AUTHOR ADDRESS: (a)ABIC Ltd., Pharm. Chem. Ind., POB 8077, Ind. Zone,  
Kiryat Nordau, Netanya\*\*Israel  
JOURNAL: International Journal for Parasitology 27 (10):p1159-1167 Oct.,  
1997  
ISSN: 0020-7519  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Transmission-blocking immunity may have great potential for use  
in the control of diseases caused by apicomplexan parasites. In this  
review I will describe our work on the application of  
transmission-blocking immunity to the control of the **Eimeria**  
parasite and compare our results to those working on  
transmission-blocking immunity against *Cryptosporidium* and *Plasmodium*.  
**Eimeria** causes the disease known as coccidiosis in domestic  
animals. Coccidiosis is particularly problematic in the chicken industry,  
mainly due to the crowded rearing conditions under which chicks are  
raised. In our work we identified, isolated and characterized 3 major  
gametocyte antigens (230 kDa, 82 kDa and 56/54 kDa) of **Eimeria**  
*maxima*. We used these native glycoproteins to **immunize** laying hens  
that, via the **egg** yolk, provide large amounts of  
transmission-blocking maternal antibodies to offspring chicks. We  
demonstrated that hatchlings from **immunized** hens shed 60-80% fewer  
oocysts (i.e., the infective stage of the life-cycle of **Eimeria**)  
than those from control hens. Such a reduction in oocyst output acts to  
significantly reduce parasite numbers in the litter of chicks raised in  
floor pens. This reduction in oocyst output is comparable to that seen  
using the most effective coccidiostat drugs and is probably sufficient to  
control coccidiosis under field conditions. Based on our results together  
with those of other groups working on transmission-blocking immunity  
against *Cryptosporidium* and *Plasmodium*, it appears that this  
immunological approach holds great promise for the control of  
apicomplexan parasites that cause diseases in both animals and man.

11/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10389204 BIOSIS NO.: 199699010349  
Biological principles of live, attenuated **vaccines**.  
AUTHOR: Shirley Martin W  
AUTHOR ADDRESS: Inst. Animal Health, Compton Lab., Compton, Nr Newbury,  
Berkshire RG20 7NN\*\*UK  
JOURNAL: Magyar Allatorvosok Lapja 51 (1):p23-29 1996  
ISSN: 0025-004X  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: Hungarian; Non-English  
SUMMARY LANGUAGE: Hungarian; English

ABSTRACT: All the live **vaccines** for controlling avian coccidiosis  
contain sporulated oocysts. The two **vaccines** introduced for the  
first time, Coccivac (Mallinckrodt Veterinary) and Immucox (Vetech

Laboratories, Canada) **vaccines** contain oocysts of coccidium populations of virulent type, i.e. the natural virulence and/or multiplication of which was not modified in no way. In spite of that, the recently developed **vaccines** - commercialized under the names Paracox (Mallinckrodt Veterinary) and Livacox (Biopharm, Research Institute of Biopharmacy and Veterinary Drugs) - contain such new populations that are characterized by significantly attenuated virulence and decreased reproductive potential. These lines were obtained from virulent parent lines by serial passages in **eggs** or by repeated selections in chickens directed onto the precocious (faster) finishing of developmental cycles. The latest method is the most effective way because it made possible to obtain attenuated populations in case of all the seven **Eimeria** species infecting chickens. As compared to the virulent parent lines, developmental cycles of precocious lines are characterized by a shorter prepatent period, and further, the oocyst production decreased significantly after having eliminated the late population(s) of schizonts. In addition to this, the intestinal wall damaging effect is significantly decreased while the **immunizing** ability remained protecting against a newer infection with the same species. Owing to the high antigenic differences observed within the *E. maxima* species, precocious lines were selected from two virulent strains and both are included into the Paracox **vaccine**. The greatest advantage of **vaccines** based on attenuated (low virulent) populations is that they ensure a more broader safety zone than the **vaccines** containing virulent parasites. The present review can be divided into the following 4 topics: Parasites, immunity and **vaccines** - introduction. The two methods used for the isolation of parasites included into the **vaccines**. Main biological characteristics of attenuated parasites (early maturing, precocious lines, illustrated by the example of *E. tenella* and *E. maxima* species). Field application of attenuated **vaccines**. WILLIAMS' and BEDRNIK's reports give a more detailed overview about the field application.

11/7/4 (Item 4 from file: 5)  
DIALOG(R) File 5: Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

09555061 BIOSIS NO.: 199598009979  
Maternal Transmission of Immunity to **Eimeria maxima**: Western Blot  
Analysis of Protective Antibodies Induced by Infection.  
AUTHOR: Smith N C(a); Wallach M; Miller C M D; Braun R; Eckert J  
AUTHOR ADDRESS: (a)Queensland Inst. Med. Res., Bancroft Cent., 300 Herston  
Rd., Brisbane, QLD 4029\*\*Australia  
JOURNAL: Infection and Immunity 62 (11):p4811-4817 1994  
ISSN: 0019-9567  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Infection of breeding hens with **Eimeria maxima** induces production of parasite-specific antibodies which are transferred, via the **egg** yolk, to hatchling chicks. These antibodies (immunoglobulin G) are highly protective, mediating up to a 97% reduction in oocyst excretion in challenged hatchlings. However, the degree of maternally derived immunity transferred by the hens to their offspring declines with increasing time after infection of the hens. This decline in immunity is directly related to declining immunoglobulin G titers. However, sera from highly protected hatchlings recognize only a very few *E. maxima* proteins on Western blots (immunoblots). In particular, a 230-kDa protein band is outstanding for its association with maternally derived immunity to *E. maxima* in hatchlings. This band was excised from a sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) preparative gel of crude merozoite protein extract. The SDS-PAGE cutout was emulsified in



Freund's adjuvant and **injected**, intramuscularly, into six breeding hens on two occasions, 2 weeks apart. **Eggs** were collected from these hens 28 to 39 days after the second **injection**, and the hatchlings from these **eggs** were challenged with 150 sporulated oocysts of *E. maxima*. Subsequent oocyst excretion in these hatchlings was, on average, 54% lower than oocyst excretion by control chicks but only 37% lower (significant at  $P < 0.05$ ) than that by chicks from hens sham **immunized** with Freund's adjuvant. The latter result is apparently due to the ability of the adjuvant to induce production of antibodies which recognize *Eimeria* spp. and thereby transfer some degree of protection to hatchlings. These experiments indicate that protective, maternally derived immunoglobulin G antibodies may be useful for the identification of putative anticoccidial **vaccine** candidates.

11/7/5 (Item 5 from file: 5)  
DIALOG(R) File 5: Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

09216022 BIOSIS NO.: 199497224392

Maternal transmission of immunity to *Eimeria* maxima: Enzyme-linked immunosorbent assay analysis of protective antibodies induced by infection.

AUTHOR: Smith Nicholas C(a); Wallach Michael; Miller Catherine M D;  
Morgenstern Ruth; Braun Richard; Eckert Johannes

AUTHOR ADDRESS: (a)Queensland Inst. Med. Res., Bancroft Centre, 300 Herston Road, Brisbane, QLD 4029\*\*Australia

JOURNAL: Infection and Immunity 62 (4):p1348-1357 1994

ISSN: 0019-9567

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

**ABSTRACT: Vaccination of broiler chickens against *Eimeria***

infection is problematic because of the need to ensure that birds are protected from the time of hatching. We have therefore investigated the feasibility of protecting hatchling broilers via maternal transfer of protective antibodies from hens to their offspring. Oral infection of broiler breeder hens with 20,000 sporulated *Eimeria* maxima oocysts caused production of antibodies which were passed into the **egg** yolk and subsequently to hatchlings. The level of specific antibodies in the yolks to unsporulated oocysts, sporulated oocysts, merozoites, and gametocytes was assessed by enzyme-linked immunosorbent assays. The levels in yolks of antibodies to all developmental stages peaked 3 to 4 weeks after infection of the hens. Groups of 10 hatchlings were challenged at 3 days of age by oral infection with 100 sporulated *E. maxima* oocysts. In the first experiment, the mean 4-day (days 6 to 9 post-infection) total number of oocysts excreted in the feces of chicks from **eggs** collected 3 weeks after infection of the hens was  $(0.6 \pm 0.4)$  times  $10^{-6}$  (mean  $\pm$  standard error) compared with  $(9.9 \pm 1.4)$  times  $10^{-6}$  for the progeny of uninfected hens, which represents a greater than 90% reduction. However, oocyst excretion by chicks from **eggs** collected 7 or 8 weeks after infection of the hens was only 47 or 68% lower than control values, reflecting declining levels of protective antibodies. In a second experiment, in which the hens were somewhat older and pretreated by intramuscular **injection** of saline in the emulsifying agent, Arlacel A, the period for which protective antibodies were transferred to hatchlings was prolonged. Thus, oocyst excretion by challenged hatchlings from **eggs** collected for an 8-week period after infection of the hens was more than 90% lower than oocyst excretion by control chicks, and even hatchlings of **eggs** collected 19 weeks after infection of the hens showed a 60% reduction in oocyst output. In both experiments, the levels of immunoglobulin G (IgG) antibodies to all developmental stages in yolks or hatchling sera were very strongly

correlated with maternally derived immunity to *E. maxima*. In contrast, parasite-specific IgM or IgA was not detectable, either in **egg** yolk or **egg** white. These results demonstrate the ability of IgG antibodies to protect against *E. maxima* in poultry, thus raising the possibility of using protective maternally derived IgG antibodies to identify potentially protective parasite antigens and indicating the feasibility of using maternal **immunization** as a means for parasite control.

11/7/6 (Item 6 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

06003228 BIOSIS NO.: 000035094591  
IN **OVO VACCINATION AGAINST EIMERIA-TENELLA**  
AUTHOR: RUFF M D; FREDERICKSEN T L; THAXTON J P; STROHLEIN D A; DANFORTH H D; GILDERSLEEVE R G  
AUTHOR ADDRESS: USDA-ARS, LIVESTOCK POULTRY SCIENCES INST., BARC-EAST, BELTSVILLE, MD. 20705.  
JOURNAL: 77TH ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION, INC. POULT SCI 67 (SUPPL. 1). 1988. 147. 1988  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

11/7/7 (Item 7 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

03246915 BIOSIS NO.: 000071060026  
**EIMERIA-NECATRIX** THE DEVELOPMENT AND CHARACTERISTICS OF AN **EGG** ADAPTED ATTENUATED LINE  
AUTHOR: SHIRLEY M W  
AUTHOR ADDRESS: HOUGHTON POULTRY RES. STATION, HOUGHTON, HUNTINGDON, CAMBS. PE17 2DA.  
JOURNAL: PARASITOLOGY 81 (3). 1980 (RECD. 1981). 525-536. 1980  
FULL JOURNAL NAME: Parasitology  
CODEN: PARAA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: Sporozoites of *E. necatrix* inoculated into the allantoic cavities of embryonating **eggs** completed their life-cycle in the chorio-allantoic membranes and produced fully viable oocysts. As a result of repeated passage in this host, an **egg**-adapted line was developed and was markedly less pathogenic for chickens than the non-passaged parent strain. Antigens capable of inducing protective immunity against challenge with low doses of the parent strain were retained, indicating a possible use for this **egg**-adapted (attenuated) line of *E. necatrix* in the **immunization** of commercially reared chickens.

11/7/8 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

10646803 Genuine Article#: 550TA Number of References: 48  
Title: Phenotypic variation among three broiler pure lines for Marek's disease, coccidiosis, and antibody response to sheep red blood cells  
Author(s): Emara MG (REPRINT) ; Lapierre RR; Greene GM; Knieriem M; Rosenberger JK; Pollock DL; Sadjadi M; Kim CD; Lillehoj HS  
Corporate Source: Univ Delaware, Dept Anim & Food Sci, Newark//DE/19717

(REPRINT); Univ Delaware, Dept Anim & Food Sci, Newark//DE/19717; Perdue Farms Inc, Salisbury//MD/21802; ARS, USDA, Parasite Biol Epidemiol Systemat Lab, Anim & Nat Resources Inst, Beltsville//MD/20705  
Journal: POULTRY SCIENCE, 2002, V81, N5 (MAY), P642-648  
ISSN: 0032-5791 Publication date: 20020500  
Publisher: POULTRY SCIENCE ASSOC INC, 1111 NORTH DUNLAP AVE, SAVOY, IL 61874-9604 USA

Language: English Document Type: ARTICLE

Abstract: To identify candidate genes, chicken lines with the most divergent phenotypes are usually crossed to generate resource mapping populations, for example, either backcrossed or F-2 populations. Linkage between the genetic marker and the phenotypic trait locus is then tested in the mapping population. As an initial step in the development of a mapping population from commercial broilers, the goal of the current research was to evaluate the phenotypic variation among three pure lines for antibody response to SRBC and in resistance to two economically important poultry diseases, Marek's disease (MD) and coccidiosis (*Eimeria acervulina*). Chicks from each line were received and separated into three experimental studies to evaluate each of their responses. In summary, broiler Line 3 had significantly lower antibody responses to SRBC immunizations compared to the other two lines, and nonvaccinated birds from Line 3 were also more susceptible to MD. With coccidiosis, the response was complex, and ranking of the lines was dependent on the age of infection, and whether it was a first or second challenge. With the first challenge, Line 1 was most susceptible at the younger age (Day 30), whereas Line 3 was susceptible at the older age (Day 58). Upon the second challenge, broiler Line 1 remained susceptible at the younger age, but Line 2 was more susceptible at the older age. Line 3 was completely resistant to the second challenge at the older age. Thus, although the broiler lines have been intensively selected for productivity and general livability, this study also demonstrates that the lines differ for immune response and disease resistance. Based on the phenotypic differences between Lines 1 and 3, they were chosen to establish a mapping population for identifying candidate genes that affect MD and coccidiosis in commercial broiler chickens.

11/7/9 (Item 2 from file: 34)  
DIALOG(R) File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

10486508 Genuine Article#: 531LY Number of References: 40  
Title: Cysteine proteinases Fas1 and Fas2 are diagnostic markers for *Fasciola hepatica* infection in alpacas (*Lama pacos*)  
Author(s): Neyra V; Chavarry E; Espinoza JR (REPRINT)  
Corporate Source: Univ Peruana Cayetano Heredia, Div Biochem & Mol Biol, POB 4314/Lima 100//Peru/ (REPRINT); Univ Peruana Cayetano Heredia, Div Biochem & Mol Biol, Lima 100//Peru/; Labs Res & Dev Sci & Technol, Mol Biotechnol Unit, Lima 100//Peru/

Journal: VETERINARY PARASITOLOGY, 2002, V105, N1 (APR 19), P21-32  
ISSN: 0304-4017 Publication date: 20020419  
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS  
Language: English Document Type: ARTICLE

Abstract: Circulating antibody against *Fasciola hepatica* antigens was determined by enzyme-linked immunosorbent assay (ELISA) and immunoelectrophoresis in alpacas naturally exposed to *F hepatica*. Serological assay parameters were established by using sera from eight infected animals and seven controls with no record of this parasitic infection. Excretory-secretory (ES-) products, Fas1- and Fas2-ELISA were used to survey 307 alpacas from a *F hepatica* endemic area in the Peruvian Andes. Seroprevalence of *F hepatica* infection varied from 56.7, 64.8 and 66.8% measured by Fas1-, Fas2- and ES-ELISA, respectively. The sensitivity for ES-ELISA was 95%, corresponding Fas1- and Fas2-ELISA sensitivity values were 90 and 95%. In this population,

7% of animals were positive for F hepatica **eggs** in faeces, other parasites detected were Trichuris sp. (40%), Nematodirus sp. (34.6%), Lamanema sp. (12.8%) and **Eimeria** sp. (11.8%). The results show that E hepatica infected animals elicit circulating antibodies against ES, Fas1 and Fas2. Fas2-ELISA may be proposed as a sensitive assay for the immunodiagnosis of fasciolosis in alpacas. (C) 2002 Elsevier Science B.V. All rights reserved.

11/7/10 (Item 3 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

06008622 Genuine Article#: XN854 Number of References: 140  
Title: Alternatives to animal experimentation in parasitology  
Author(s): Eckert J (REPRINT)  
Corporate Source: UNIV ZURICH, INST PARASITOL, WINTERTHURERSTR 266A/CH-8057 ZURICH//SWITZERLAND/ (REPRINT)  
Journal: VETERINARY PARASITOLOGY, 1997, V71, N2-3 (JUL 31), P99-120  
ISSN: 0304-4017 Publication date: 19970731  
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS  
Language: English Document Type: ARTICLE  
Abstract: In parasitology, several measures can contribute to the aims of 3R (Reduction, Refinement and Replacement of animal experimentation). These include legal regulations, international guidelines for evaluating antiparasitic drugs, the refinement of animal models for parasitic infections, cryopreservation and cultivation of parasites in vitro or in chicken **eggs**, the maintenance of arthropods by artificial feeding, and the use of immunological and molecular in vitro techniques (e.g. the production of recombinant antigens for **vaccines**). Considerable progress has been achieved in the development of alternative techniques but both their standardisation and validation are not far advanced. A wider acceptance and use of alternative methods will only be achieved if research can offer economic alternatives that are as effective and reliable as animal experiments. Great efforts are needed for further progress. (C) 1997 Elsevier Science B.V.

11/7/11 (Item 4 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

04112138 Genuine Article#: RF010 Number of References: 25  
Title: POTENTIAL CONTROL OF CHICKEN COCCIDIOSIS BY MATERNAL **IMMUNIZATION**  
Author(s): WALLACH M; SMITH NC; BRAUN R; ECKERT J  
Corporate Source: ABIC LTD PHARMACEUT & CHEM IND, IND ZONE, KIRYAT NORDAU, POB 8077/NETANYA//ISRAEL/; QUEENSLAND INST MED RES, BANCROFT CTR/BRISBANE/QLD 4029/AUSTRALIA/; UNIV BERN, INST ALLGEMEINE MIKROBIOL/CH-3012 BERN//SWITZERLAND/; UNIV ZURICH, INST PARASITOL/CH-8057 ZURICH//SWITZERLAND/  
Journal: PARASITOLOGY TODAY, 1995, V11, N7 (JUL), P262-265  
ISSN: 0169-4758  
Language: ENGLISH Document Type: ARTICLE  
Abstract: The control of coccidiosis by maternal **immunization** represents an important potential strategy in the fight against this serious veterinary health problem. In this short review, Michael Wallach, Nicholas Smith, Richard Braun and Johannes Eckert present a summary of results obtained in work carried out in several laboratories in Israel and Switzerland on the fundamental aspects of maternal immunity and its implications for **vaccine** development against coccidiosis in chickens.

11/7/12 (Item 1 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

04152221 CAB Accession Number: 20023000206  
Live coccidiosis **vaccine** by in ovo.  
World Poultry vol. 17 (12): p.24  
Publication Year: 2001  
ISSN: 1388-3119 --  
Language: English  
Document Type: Journal article

11/7/13 (Item 2 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03824735 CAB Accession Number: 992217566  
**Vaccination** of broiler parents against coccidiosis - a field trial.  
Original Title: **Vaccination** av slaktkycklingforaldrar mot koccidios - en faltstudie.  
Lunden, A.; Thebo, P.  
Avdelningen for parasitologi, Statens veterinarmedicinska anstalt och Sveriges lantbruks-universitet, Box 7073, 750 07 Uppsala, Sweden.  
Svensk Veterinartidning vol. 51 (14): p.701-705  
Publication Year: 1999  
ISSN: 0346-2250 --  
Language: Swedish Summary Language: english  
Document Type: Journal article

**Vaccination** with Paracox, a live **vaccine**, was evaluated under field conditions in Sweden between 1996 and 1997. The study included 23 flocks of commercially reared broiler breeders. The farms had previously reared the birds without any in-feed coccidiostats. If signs of coccidiosis were observed the flock was medicated with an anticoccidial drug. To compare **vaccination** with this strategy, every second rearing flock was **vaccinated** during the period. The **vaccine** was given via drinking water when the chickens were one-week-old. Coccidiosis was not diagnosed in any of the 11 **vaccinated** flocks while 5 of the 12 unvaccinated flocks showed signs of coccidiosis. Analysis of litter samples collected at regular intervals during the rearing period revealed that oocyst levels were generally lower in **vaccinated** flocks than in the control flocks. Mortality of male chickens during the rearing period showed no difference between **vaccinated** an unvaccinated flocks, while it was significantly higher in **vaccinated** female chickens than in unvaccinated. There was no significant difference in production (eggs/hen) or mortality during the laying period between **vaccinated** and unvaccinated flocks. It is concluded that **vaccination** effectively protected the birds from outbreaks of clinical coccidiosis during the rearing period and it was at least as effective as the previously used strategy of natural **immunization** and drug therapy to prevent losses caused by coccidial infections during the laying period. 6 ref.

11/7/14 (Item 3 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

03525272 CAB Accession Number: 980802742  
A trial for **immunization** of chicks against caecal coccidiosis using irradiated **Eimeria tenella** oocysts: pathological evaluation.  
Fetaih, H. A.; Aly, S. M.  
Department of Pathology and Clinical Pathology, Faculty of Veterinary

Medicine, Suez Canal University, Egypt.

Egyptian Journal of Comparative Pathology and Clinical Pathology vol.  
10 (1): p.133-146

Publication Year: 1997 --

Language: English

Document Type: Journal article

The effect of different doses of irradiation (5, 10, 15, 20, 25 and 30 Kr of gamma rays) on the immunogenicity of 20x103 **Eimeria tenella** oocysts was investigated in 160 Hy-line egg-type chicks aged 2 weeks. The pathological evaluation of the immune response was based on calculating the gross lesions and histopathological scores as well as the grades of coccidial stages in tissue. Following challenge by 50x103 viable oocysts, the best immunogenicity was found to be achieved by exposure of oocysts to 20 Kr before inoculation, as indicated by the low scores obtained. A second experiment was carried out to study the effect of different numbers of irradiated oocysts exposed to 20 Kr. Following challenge by 50x103 viable oocysts, the best **immunizing** effect was achieved by inoculation of 20x103 irradiated oocysts per chick. 35 ref.

11/7/15 (Item 4 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

02948148 CAB Accession Number: 950800342

Maternal transfer of antibodies induced by infection with **Eimeria maxima** partially protects chickens against challenge with **Eimeria tenella**.

Smith, N. C.; Wallach, M.; Petracca, M.; Braun, R.; Eckert, J.

Institut fur Parasitologie, Universitat Zurich, Winterthurerstrasse  
266a, CH-8057, Zurich, Switzerland.

Parasitology vol. 109 (5): p.551-557

Publication Year: 1994

ISSN: 0031-1820 --

Language: English

Document Type: Journal article

Infection of breeding hens with **Eimeria maxima** induces production of **Eimeria**-specific IgG antibodies which are transferred to hatchlings via the egg yolk and confer a high degree of maternal immunity against homologous challenge and partial immunity to infection with another important species, **Eimeria tenella**. As an example, in an experiment using hatchlings from eggs collected between days 28 and 39 after infection of the hens with 20 000 sporulated *E. maxima* oocysts, control chicks (challenged with 100 sporulated oocysts) excreted 6.8 plus or minus 1.2 million (mean plus or minus S.E., n = 10) or 5.8 plus or minus 1.2 million (n = 8) oocysts of *E. maxima* or *E. tenella*, respectively, compared to 0.9 plus or minus 0.4 million (n = 5) *E. maxima* oocysts or 2.2 plus or minus 0.4 million (n = 9) *E. tenella* oocysts excreted by hatchlings of infected hens. This represents an 87% reduction in oocyst excretion with regard to *E. maxima* and a 62% reduction in oocyst excretion with regard to *E. tenella* in the progeny of the infected hens. In another experiment, eggs were collected from days 28 to 37 and again from days 114 to 123 after infection of the hens with *E. maxima* and hatchling oocyst excretion rates were 82% and 62%, respectively, reduced for *E. maxima* and 43% and 41%, respectively, reduced for *E. tenella* in the progeny of hens infected with *E. maxima* compared to the progeny of uninfected hens. ELISA and Western blot analyses of maternally-derived IgG revealed a high degree of cross-reactivity to antigens of *E. maxima* and *E. tenella*. Thus, maternally-derived, IgG-mediated cross-resistance to different species of **Eimeria** occurs in the chicken, most likely as a result of cross-recognition of conserved epitopes or proteins. 15 ref.

11/7/16 (Item 5 from file: 50)

DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02821738 CAB Accession Number: 940801938

Preparation of a culture of coccidia for immunoprophylaxis of chickens and its testing in the field.

Kontrimavichyus, V.; Arnastauskene, T.; Skonsmanas, I.; Sruoga, A.; Paulauskas, A.; Butkauskas, D.; Drebitskene, G.

Institute of Ecology, Vilnius, Lithuania.

Biologija (No. 1.): p.86-87

Publication Year: 1993

Sovremennye Problemy Parazitologii v Stranakh Baltii. (Proceedings of the 12th Baltic Parasitological Conference) --

Language: Russian

Document Type: Conference paper; Journal article

A **vaccine** containing **Eimeria tenella**, **E. acervulina** or **E. maxima** oocysts at the ratio of 1.5:15:0.5 in a 2% solution of potassium bichromate was tested in 2 equal groups of chicks totalling 30 770 chicks at a factory farm in Lithuania. The experimental group received the **vaccine** in food at the age of 10 days. The results suggest that **immunization** of **egg**-laying breeds raised in cages where coccidiosis does not cause serious problems, is justified because it reduced the number of starvelings and stimulated non-specific immunity. 3 ref.

11/7/17 (Item 6 from file: 50)

DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02539242 CAB Accession Number: 920877668

Effect of **Eimeria nieschulzi** on **Nippostrongylus brasiliensis**-induced IgE.

Sambrano, G. R.; Mayberry, L. F.; Bristol, J. R.

L.F. Mayberry, Department of Biological Sciences, The University of Texas at El Paso, El Paso, TX 79968-0519, USA.

Parasitology Research vol. 78 (2): p.172-174

Publication Year: 1992

ISSN: 0044-3255 --

Language: English

Document Type: Journal article

Twelve male Sprague-Dawley rats were inoculated orally with  $2.5 \times 10^5$  sporulated oocysts of *E. nieschulzi* and 6 were challenged with the same dose on day 15 pi. 27 rats were infected sc with  $2 \times 10^3$  L3 of *N. brasiliensis* and then challenged at 30 days pi; 12 of these were also inoculated with  $2.5 \times 10^5$  oocysts of *E. nieschulzi* 8 days pi. Sera were tested for parasitic-specific reaginic antibody by passive cutaneous anaphylaxis (PCA). In a 2nd experiment, 3 groups of 3 rats were **immunized** ip with chicken-**egg** albumin. After 20 days, 2 groups were inoculated with  $2 \times 10^3$  L3 of *N. brasiliensis*; 4 days later, rats in one of these groups were inoculated with  $2.5 \times 10^5$  sporulated oocysts of *E. nieschulzi*. On day 12 pi with *N. brasiliensis*, sera were collected and tested against **egg** albumin by PCA. The results of the 2 experiments showed that *E. nieschulzi* did not exert a significant suppressive effect on *N. brasiliensis*-induced IgE. It is suggested that the suppressive effect of *E. nieschulzi* is not the result of IgE depression, but instead results from the suppression of an unknown alternative component that is important to self-cure. 10 ref.

11/7/18 (Item 7 from file: 50)

DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

02404838 CAB Accession Number: 910871209

In **ovo** administration of a potential recombinant coccidial antigen **vaccine** in poultry.

Fredericksen, T. L.; Thaxton, J. P.; Gildersleeve, R. P.; Rowe, D. G.; Ruff, M. D.; Strohle, D. A.; Danforth, H. D.

EMBREX, Inc., PO Box 13989, Research Triangle Park, NC 27709, USA.

Conference Title: Coccidia and intestinal coccidiomorphs. Proceedings of the 5th International Coccidiosis Conference. Tours (France), 17-20 October 1989.

p.655-660

Publication Year: 1989

Les Colloques de l'INRA, No. 49

Editors: Yvore, P.

Publisher: INRA Service des Publications -- Versailles, France

ISBN: 2-7380-0164-5

Language: English

Document Type: Conference paper

EMBREX, Inc., has developed an **egg injection** machine (INOVOJECT) that simultaneously **vaccinates** and transfers chicken **eggs** at the rate of 20 000 per hour. The first commercial products will include viral **vaccines** for Marek's disease, infectious bursal disease, Newcastle disease, and infectious bronchitis. In addition, a recombinant coccidial antigen (3264) derived from **Eimeria tenella** and cloned in E. coli is being developed as a potential in **ovo vaccine** for coccidiosis in broiler chickens. This antigen, when given to embryos at day 18 of embryonation, evoked a significant humoral immune response and reduced the severity of caecal lesions in chicks orally challenged with E. **tenella** oocysts. In **ovo vaccination** with an appropriate recombinant coccidiosis antigen holds promise as an efficacious method of preventing coccidiosis. 12 ref.

11/7/19 (Item 8 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

01320877 CAB Accession Number: 830808323

Attenuation of **Eimeria mivati** (= **mitis**) by selection for precocious development.

McDonald, V.; Ballingall, S.

Houghton Poultry Res. Sta., Houghton, Huntingdon, Cambs. PE17 2DA, UK.

Parasitology vol. 86 (3): p.371-379

Publication Year: 1983

ISSN: 0031-1820 --

Language: English

Document Type: Journal article

By selection for early development of oocysts during serial passage through chickens the pre-patent period of the Houghton (H) strain of E. **mivati** (= **mitis**) was reduced by over 20 h. The precocious parasite was less pathogenic than the H strain and had a reduced reproductive potential. Chicks inoculated with the precocious parasite were protected against challenge with the H strain. The pathogenicity, immunogenicity and reproduction of precocious E. **mivati** and an attenuated **egg**-adapted line of E. **mivati** were compared. Although the precocious parasite produced more oocysts than the **egg**-adapted parasite in chickens, neither parasite induced loss of weight and both were immunogenic. (AS). 14 ref.

11/7/20 (Item 9 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

01178024 CAB Accession Number: 820897153

An **egg**-adapted (attenuated) line of **Eimeria necatrix**:

further studies on its reproduction, pathogenicity and immunogenicity.

Shirley, M. W.; Bellatti, M. A.; Millard, B. J.



Houghton Poultry Res. Stn., Houghton, Huntingdon, Cambs PE 17 2DA, UK.  
Parasitology vol. 84 (2): p.215-226  
Publication Year: 1982  
ISSN: 0031-1820 --  
Language: English  
Document Type: Journal article

An **egg**-adapted line of *E. necatrix* has been passaged 40 times in the chorio-allantoic membranes of embryonated **eggs**. Between the 21st and 40th passage in **eggs** the parasite was subjected to a selection for precocious development, and its pre-patent period in this host is now 123-125 h. The parasite continued to adapt to the chorio-allantoic membrane throughout the period of the passages and its development in this tissue caused the growth of the host embryo to be markedly stunted. In chickens, both the reproduction and the pathogenicity of the **egg**-adapted line decreased with increasing numbers of passages in **eggs**. A comparison of the immunogenicity of the **egg**-adapted line and its parent strain in chickens kept on litter showed that substantially more oocysts of the **egg**-adapted line had to be given to induce complete protection. (AS). 13 ref.

11/7/21 (Item 10 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01086384 CAB Accession Number: 802259894

Incorporation, persistence and transmission of Newcastle disease virus in various species of parasite (*Eimeria*, *Ascaridia galli*).

Original Title: Dosadasnji rezultati ispitivanja mogucnosti inkorporisanja, odrzavanja i prenosnja virusa Newcastlea preko nekih vrsta parazita.

Sibalic, S.; Mihajlovic, B.; Tomanovic, B.; Asanin, R.

Vet. Fak., Univ., Belgrade, Yugoslavia.

Veterinaria, Yugoslavia vol. 29 (1/2): p.241-244

Publication Year: 1980

English summary pp.400-401 --

Language: Serbo-Croatian

Document Type: Journal article

It was shown that Newcastle disease virus (NDV) can be present in coccidial oocysts excreted by chicks infected with Newcastle disease, and NDV incorporated in *Eimeria tenella* oocysts can be transmitted to a receptive individual perorally, parenterally or by intraperitoneal deposition. Only 24 hours after infection with NDV it was shown to be present in oocysts. NDV survived for up to 8 months in oocysts of *E. tenella* stored in a refrigerator at 3-5 deg C. The virus was also reisolated from oocysts of *E. tenella*, excreted by chicks vaccinated with T.B. Mukteswar strain, Satto strain NDV can be incorporated in *E. necatrix* during endogenous development in a host organism also infected with NDV. *E. necatrix* oocysts containing NDV were administered to susceptible birds perorally or parenterally, and these birds showed symptoms of Newcastle disease with death, or subsequent immunity to lethal NDV doses. The incorporation of NDV into larval (21 day old) and mature (45 days old) forms of *Ascaridia galli* and in **eggs** of *A. galli* was also demonstrated. 8 ref.

11/7/22 (Item 11 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

01036371 CAB Accession Number: 810878683

Annual report 1979/80.

Britain, Agricultural Research Council

v + 126 pp.

Publication Year: 1980

Protozoology pp. 48-49, 50-51

Publisher: Her Majesty's Stationery Office. -- London, UK

Language: English

Document Type: Miscellaneous

At the Houghton Poultry Research Station an attenuated line of **Eimeria necatrix** has been developed by serial passage in embryonating hens **eggs**. The parent chicken-maintained strain from which the attenuated, **egg**-adapted line was developed caused severe disease (in one test 60% of chickens died following inoculation with 50 000 **E. necatrix**). Severe pathological changes in the intestine and loss of body weight were noted. In contrast, the attenuated line passaged 36 times in **eggs** caused no deaths even when inoculations of 200 000 **E. necatrix** were given. Only minor pathological changes and no effect on weight gain were observed. Inoculation of attenuated line, passaged 22 times in **eggs**, produced immunity against challenge with the virulent parent strain. There were no deaths and weight gains were similar to those of control chickens. At the Moredun Institute, Edinburgh, Scotland, toxoplasmosis has been found to be associated with immunosuppression. 60% of sheep infected with *Toxoplasma gondii* and 7 days later infected with louping-ill virus died; with the virus alone 30% of sheep died and with *T. gondii* alone all animals survived. Sheep with toxoplasmosis did not respond well to commercial louping-ill virus **vaccine** or to enzootic abortion **vaccine**. ADDITIONAL ABSTRACT:

A more rapid approach to screening breeding material for resistance to potato cyst nematodes (*Globodera* spp.) has been adopted by the Scottish Plant Breeding Station. The technique involves planting small tubers or eye scoops in small transparent containers which are inoculated with a suspension of nematode **eggs** and placed for 6 to 8 weeks in the dark at 20 deg C. Visible females on the root system are counted through the container wall.

11/7/23 (Item 12 from file: 50)

DIALOG(R)File 50:CAB Abstracts

(c) 2002 CAB International. All rts. reserv.

00104780 CAB Accession Number: 732206147

Scientific report of the Division of Parasitology for the year 1971.

Indian Veterinary Research Institute

28pp.

Publication Year: 1972

Publisher: -- n Veterinary Research Institute, Izatnagar UP., India

Language: English

Document Type: Annual report

In Northern India the following genera of ticks were recorded: *Ixodes*, *Haemaphysalis*, *Boophilus*, *Rhipicephalus*, *Dermacentor* and *Hyalomma*. TEPA was used successfully as chemosterilant in *Stomoxys calcitrans* and TEPA plus radiation in *Argus persicus*. In pigs, Distodin and tetrachloroethylene possessed anthelmintic activity against *Fasciola buski*. Irradiated and non-irradiated **eggs** were used for **immunization** against *Ascaridia galli* infection in chicks. The indirect HA titre was 1:5 to 1:15 in the first week of **vaccination** and rose to 1:320 in the fourth week; the titre was lower in birds given irradiated **eggs**. The intensity of *Bunostomum trigonocephalum* infection was investigated in sheep and goats. Guinea-pigs and mice were infected with the larvae of this worm. The infection rate of *Haemonchus contortus* in sheep and goats varied between 95 and 98%. Rats, mice and guinea-pigs were not suitable hosts for this parasite. *Fasciola gigantica* infection could not be established in 608 *Lymnaea auricularis* examined but 50% buffaloes and 40% cattle and goats were infected. Oocysts of **Eimeria necatrix** did not develop resistance to Codrinol up to tenth passage level and also did not change their sensitivity either to sulphaquinoxaline or amprolium. The incidence of *Dictyocaulus filaria* in sheep farms was 29.4%; animals **vaccinated** with irradiated **vaccine** between two and 18 months of age and again 28 days later

showed an incidence of 3.5% against 25.8% in controls; the **vaccinated** animals also showed a higher body weight. Dimethoate was most effective against *Stomoxys calcitrans* followed by coumaphos, diazinon, sevin, BHC, sumithion, DDT and heptachlor. In birds, the incidence of *Ascaris galli* was 42.5%, *Heterakis gallinae* 64.5% and cestodes 33%. Thiabendazole, tetramisole and phenothiazine were highly effective against mature and immature *H. gallinae*. The **eggs** of this worm irradiated at 15 or 20 kr were highly immunogenic. Other subjects studied included; irradiated **vaccine** against *Fasciola gigantica* infection; immunodiagnosis of cysticercosis in pigs and coenurosis in sheep and goats; immunology of *Schistosoma incognitum*; therapeutics of poultry coccidia.

11/7/24 (Item 13 from file: 50)  
DIALOG(R)File 50:CAB Abstracts  
(c) 2002 CAB International. All rts. reserv.

00102741 CAB Accession Number: 722203535  
Immunity to coccidiosis: maternal transfer in **Eimeria** maxima infections.

Rose, M. E.  
Poultry Res. Stn., Houghton, Huntingdon.  
Parasitology vol. 65 (Part 2): p.273-282  
Publication Year: 1972  
ISSN: 0031-1820 --  
Language: English  
Document Type: Journal article

Chicks hatched from **eggs** laid in the third and fourth weeks after inoculation of the hens with oocysts of *E. maxima* excreted fewer oocysts in response to a challenge inoculum of *E. maxima* given at 1 week of age than controls hatched from the **eggs** of uninfected hens. A gamma-livetin fraction prepared from the yolks of **eggs** laid by hens 14-22 days after a single inoculation of oocysts of *E. maxima* and **injected** subcutaneously into 2-week-old chicks caused a similar reduction in oocyst yield from a challenge inoculum. The serum of the donor hens, obtained at the corresponding time intervals was protective. These results confirm that immunity to infection with *E. maxima* is, at least in part, mediated by humoral antibodies.

11/7/25 (Item 1 from file: 76)  
DIALOG(R)File 76:Life Sciences Collection  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

02131717 4039152  
Method of reducing the output of **Eimeria** oocysts from a newborn chick  
CHILWALNER  
PATENT NUMBER: US 5496550  
(1996)  
DOCUMENT TYPE: Patent LANGUAGE: ENGLISH  
SUBFILE: Microbiology Abstracts C: Algology, Mycology & Protozoology

This invention provides a method of reducing the output of **Eimeria** oocysts from a newborn chick which comprises administering to a laying hen at a suitable time prior to the hen laying a fertilized **egg** an amount of native or recombinant antigenic protein present in gametocytes of the **Eimeria** spp. effective to induce in the hen an immune response conferring protection via maternal immunity against infection or transmission by the **Eimeria** spp. in the offspring chick.

11/7/26 (Item 2 from file: 76)  
DIALOG(R)File 76:Life Sciences Collection  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

01805548 3582428

In vitro activity of the human neutrophil cathepsin G on **Eimeria tenella** sporozoites

Guyonnet, V.; Johnson, J.K.; Bangalore, N.; Travis, J.; Long, P.L.

Dep. Poult. Sci., Univ. Georgia, Athens, GA 30602, USA

J. PARASITOL. vol. 77, no. 5, pp. 775-779 (1991)

ISSN: 0022-3395

DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH

SUBFILE: Microbiology Abstracts C: Algology, Mycology & Protozoology

The role of human neutrophil cathepsin G (Cat G) on **Eimeria tenella** sporozoites was studied in vitro. Sporozoites were incubated for 2 hr at 37 C in PO sub(4) buffer, 0.9% NaCl (PBS), pH 7.6 in the presence of Cat G (50  $\mu$ g/ml), diisopropyl fluorophosphate-inhibited Cat G (DFP-Cat G) (50  $\mu$ g/ml) or PBS alone, prior to being inoculated into embryonated **eggs**. As judged by oocyst production on day 7 postinoculation, embryo mortality and the hemorrhage scores, both Cat G and DFP-Cat G demonstrated anticoccidial activity greater activity was obtained with the DFP-Cat G. Sporozoites were exposed also to increasing concentrations of native and trypsin-digested DFP-Cat G (0-100  $\mu$ g/ml) under the same conditions. Significant protection (37% and 49% for native and digested DFP-Cat G, respectively) was obtained with a low concentration (5  $\mu$ g/ml), and higher concentrations resulted in 70% and 84% protection, respectively. The primary bactericidal domain of Cat G, the HPQYNQR peptide, at 3 concentrations (25, 50, and 100  $\mu$ g/ml), reduced the oocyst production by 46%, 16%, and 15%, respectively. The anticoccidial activity of Cat G may involve a peptide fragment different from the antimicrobial domain of the enzyme.

11/7/27 (Item 3 from file: 76)  
DIALOG(R)File 76:Life Sciences Collection  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

00962076 1354378

Some observations on the biology of five strains of **Eimeria**

**necatrix**.

Shirley, M.W.

Houghton Poult. Res. Stn., Houghton, Huntingdon, Cambridge PE17 2DA, UK

Z. PARASITENKD. vol. 71, no. 3, pp. 287-295 (1985.)

DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH

SUBFILE: Microbiology Abstracts Section C: Algology, Mycology and Protozoology

Five laboratory strains of **Eimeria necatrix** were characterised with regard to the size of their oocysts, pathogenicity, reproduction, cross-immunity, ability to grow in embryonated **eggs**, and electrophoretic variation of enzymes. Three strains were highly pathogenic whilst two caused only few deaths and milder changes to the mean body weight gains of infected chickens. Cross-immunity was incomplete judged by scores of lesions after heterologous challenge, and electrophoretic variation of the enzymes lactate dehydrogenase and isocitrate dehydrogenase from oocysts of the five strains was also found. All the strains completed their life cycle in embryonated **eggs** but only a few oocytes were recovered.

11/7/28 (Item 4 from file: 76)  
DIALOG(R)File 76:Life Sciences Collection  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

00688437 0565234

Antibodies to coccidia: Detection by the enzyme-linked immunosorbent assay (ELISA).

Rose, M.E.; Mockett, A.P.A.  
Houghton Poultry Res. Stn., Houghton, Huntingdon, Cambs. PE17 2DA, UK  
PARASITE IMMUNOL. vol. 5, no. 5, pp. 479-489 (1983.)  
DOCUMENT TYPE: Journal article LANGUAGE: ENGLISH  
SUBFILE: Microbiology Abstracts Section C: Algology, Mycology and  
Protozoology

The ELISA test was used for the detection of antibodies to coccidia in the serum and/or egg yolk of chickens infected with **Eimeria acervulina**, **E. maxima** or **E. tenella** and in the serum of rats infected with **E. nieschulzi**. Antigens prepared from different developmental stages of the parasite were tested and the cross-reaction between different species of **Eimeria** were examined. The variability in cross-reactivity of different species and the advantages and possible applications of the test are discussed.

11/7/29 (Item 1 from file: 77)  
DIALOG(R)File 77:Conference Papers Index  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

4614668  
Supplier Accession Number: 01-06983 V29N06  
Movement of oocysts within chicken embryos after in ovo vaccination with **Eimeria maxima**  
Weber, F.H.; Farrand, M.; LeMay, M.A.; Lewis, D.O.; Genteman, K.C.; Evans, N.A.  
8th International Coccidiosis Conference 0005692 Cairns (Australia)  
9-13 Jul 2001  
Molecular Parasitology Unit (University of Technology, Sydney),  
Australian Society for Parasitology  
University of Technology, Sydney, Department of Cell and Molecular  
Biology, Westbourne St, Gore Hill NSW 2065, Australia; phone:  
61-2-9514-4063; fax: 61-2-9514-4026. Poster Paper  
Languages: ENGLISH

11/7/30 (Item 2 from file: 77)  
DIALOG(R)File 77:Conference Papers Index  
(c) 2002 Cambridge Sci Abs. All rts. reserv.

4614598  
Supplier Accession Number: 01-06976 V29N06  
Efficacy of in ovo vaccination with single and mixed species of live **Eimeria** oocysts  
LeMay, M.A.; Genteman, K.C.; Weber, F.H.; Lewis, D.O.; Evans, N.A.  
8th International Coccidiosis Conference 0005692 Cairns (Australia)  
9-13 Jul 2001  
Molecular Parasitology Unit (University of Technology, Sydney),  
Australian Society for Parasitology  
University of Technology, Sydney, Department of Cell and Molecular  
Biology, Westbourne St, Gore Hill NSW 2065, Australia; phone:  
61-2-9514-4063; fax: 61-2-9514-4026  
Languages: ENGLISH

11/7/31 (Item 1 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

12782530 21454090 PMID: 11567773  
Adjuvant effects of IL-1beta, IL-2, IL-8, IL-15, IFN-alpha, IFN-gamma  
TGF-beta4 and lymphotactin on DNA vaccination against **Eimeria acervulina**.  
Min W; Lillehoj H S; Burnside J; Weining K C; Staeheli P; Zhu J J  
Parasite Biology, Epidemiology, Systematics Laboratory, Animal and

Natural Resources Institute, BARC-East, Building 1040, US Department of Agriculture, Beltsville, MD 20705, USA.

Vaccine (England) Oct 12 2001, 20 (1-2) p267-74, ISSN 0264-410X  
Journal Code: 8406899

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Eight chicken cytokine genes (IL-1beta, IL-2, IL-8, IL-15, IFN-alpha, IFN-gamma, TGF-beta4, lymphotactin) were evaluated for their adjuvant effect on a suboptimal dose of an **Eimeria** DNA vaccine carrying the 3-1E parasite gene (pcDNA3-1E). Chickens were given two subcutaneous injections with 50 microg of the pcDNA3-1E vaccine plus a cytokine expression plasmid 2 weeks apart and challenged with **Eimeria acervulina** 1 week later. IFN-alpha (1 microg) or 10 microg of lymphotactin expressing plasmids, when given simultaneously with the pcDNA3-1E vaccine, significantly protected against body weight loss induced by **E. acervulina**. Parasite replication was significantly reduced in chickens given the pcDNA3-1E vaccine along with 10 microg of the IL-8, lymphotactin, IFN-gamma, IL-15, TGF-beta4, or IL-1beta plasmids compared with chickens given the pcDNA3-1E vaccine alone. Flow cytometric analysis of duodenum intraepithelial lymphocytes showed chickens that received the pcDNA3-1E vaccine simultaneously with the IL-8 or IL-15 genes had significantly increased CD3+ cells compared with vaccination using pcDNA3-1E alone or in combination with the other cytokine genes tested. These results indicate that the type and the dose of cytokine genes injected into chickens influence the quality of the local immune response to DNA vaccination against coccidiosis.

Record Date Created: 20010924

11/7/32 (Item 2 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

12718144 21525165 PMID: 11666150

A novel method for counting eimerian oocysts at very low concentrations in aqueous suspensions.

Williams R B; Marshall J A; Catchpole J

Schering-Plough Animal Health, Breakspear Road South, Harefield, Uxbridge, Middlesex UB9 6LS, UK. ray.williams@spcorp.com

Research in veterinary science (England) Aug 2001, 71 (1) p67-71,  
ISSN 0034-5288 Journal Code: 0401300

Document type: Journal Article; Validation Studies

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

A novel method for counting eimerian oocysts in samples of drinking water has been developed to fulfil the need for monitoring the delivery of very low concentrations of live anticoccidial vaccines to poultry via pipeline nipple-drinker systems. Advantages of the method are the ease of sample collection and processing, high degrees of accuracy and precision, and a sensitivity of one oocyst ml<sup>-1</sup>. Results of a validation test are presented, with a protocol for the method and notes on its use. The coefficient of variation (CoV) of 10 sets of oocyst counts with nominal means of 10 to 160 oocysts ml<sup>-1</sup> ranged from about 16 per cent down to 6 per cent. The recovery efficiency for all 100 validation counts averaged 100.2 per cent with a range of 70-130 per cent. A practical example of field use of the method is given, including a modification to decrease the time taken for counting. In this case, when oocysts were pumped around a pipeline circuit of 129 m for 2.5 hours, the CoV of a mean of 112 oocysts ml<sup>-1</sup> (n = 10) was 12.4 per cent. Copyright 2001 Harcourt Publishers Ltd.

Record Date Created: 20011022

11/7/33 (Item 3 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

12611613 21563210 PMID: 11706844

**Vaccines** against the avian enteropathogens **Eimeria**,  
**Cryptosporidium** and **Salmonella**.

Lillehoj E P; Yun C H; Lillehoj H S

Department of Pharmaceutical Sciences, School of Pharmacy, University of  
Maryland, Baltimore 21201, USA.

Anim Health Res Rev (England) Jun 2000, 1 (1) p47-65, ISSN  
1466-2523 Journal Code: 101083072

Document type: Journal Article; Review; Review, Academic

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The worldwide poultry industry provides a substantial proportion of the nutritional requirement of the human population. To keep pace with the increasing demand for the high-quality, low-cost protein source that poultry provides, intensive rearing practices have been developed within the past few decades. For example, chickens are housed routinely in crowded environments under adverse conditions, and genetic strains have been selected for rapid growth, high protein-to-fat content and superior egg-laying characteristics. A major negative consequence of these practices has been an increase in the incidence of diseases. Enteric diseases in particular have emerged as a major problem threatening the future viability of the poultry industry. A variety of methods have been used to combat avian diseases in the commercial setting, including improved farm management practices, the use of antibiotic drugs, the selection of disease-resistant strains of chickens, and the manipulation of the chicken's immune system. In the latter category, the development of **vaccines** against the major avian diseases has become a priority in the poultry industry. This review will highlight recent progress in **vaccine** development against three major avian enteric pathogens:

**Eimeria**, **Cryptosporidium** and **Salmonella**. (155 Refs.)

Record Date Created: 20011114

11/7/34 (Item 4 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

10847852 20401423 PMID: 10945126

A gel delivery system for coccidiosis **vaccine**: uniformity of  
distribution of oocysts.

Dasgupta T; Lee E H

Vetech Laboratories Inc., Guelph, Ontario.

Canadian veterinary journal. La revue veterinaire canadienne (CANADA)  
Aug 2000, 41 (8) p613-6, ISSN 0008-5286 Journal Code: 0004653

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

A patented gel delivery system being used to deliver coccidiosis **vaccine** to poultry hatchlings is assessed. For effective **vaccination**, the coccidial oocysts must be uniformly suspended before exposure to birds. The uniformity of distribution within the gel was evaluated by incorporating a culture of chicken gut flora into gel sausages, placing sections of the sausage on culture plates, determining the appearance and distribution of bacterial colonies on culture plates after incubation, and verifying by cell counts. The uniformity of distribution of similarly prepared coccidial oocysts was verified by infecting birds with 40,000 **Eimeria tenella** oocysts delivered via the gel. Gel-inoculated birds were compared with control birds inoculated PO with 40,000 oocysts suspended in water by using cecal lesion scores. Both the appearance and colony counts of chicken gut flora from the gel were uniform. The standard deviation in the lesion scores for the gel-inoculated group and the water-inoculated groups were 0.51 and 0.69,

respectively. The results indicate that a gel delivery system can provide uniform distribution of live organisms and **vaccine** agents to birds.

Record Date Created: 20001004

11/7/35 (Item 5 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

10354500 99346746 PMID: 10418192

Comparison between a live, attenuated anticoccidial **vaccine** and an anticoccidial ionophore, on performance of broilers raised with or without a growth promoter, in an initially **Eimeria**-free environment.

Waldenstedt L; Lunden A; Elwinger K; Thebo P; Ugglä A

Department of Animal Nutrition and Management, Swedish University of Agricultural Sciences, Uppsala, Sweden. Lotta.Waldenstedt@huv.slu.se

Acta veterinaria Scandinavica (DENMARK) 1999, 40 (1) p11-21, ISSN 0044-605X Journal Code: 0370400

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

An experiment was carried out to study the effects of **vaccination** with Paracox, a live, attenuated **vaccine** against avian coccidiosis, on broilers isolated from extraneous **Eimeria** parasites. The study involved 3200 broiler chickens raised in floor pens similar to commercial conditions, but in an initially **Eimeria**-free environment. Forty percent of the chickens were **vaccinated** at 3 days of age and given either a basal unmedicated feed or a feed supplemented with the feed antibiotic virginiamycin. Unvaccinated birds were given either the basal feed or feed supplemented either with virginiamycin or the anticoccidial ionophore narasin. At slaughter at 36 days of age **vaccinated** birds had a lower live weight than non-**vaccinated** birds. The difference was 4.6% in unmedicated, and 6.0% in virginiamycin medicated chickens. Feed conversion ratio at slaughter was 2.5% higher for unmedicated **vaccinated** birds, and 1.3% higher for virginiamycin medicated **vaccinated** birds, compared to respective non-**vaccinated** groups. There was no significant difference in overall performance of unvaccinated birds given narasin as compared to virginiamycin. At 10 days post **vaccination** **vaccinated** birds had a higher number of Clostridium perfringens in the caeca, but there was no difference thereafter. Throughout the experiment, caecal clostridial counts were considerably higher in **vaccinated** unmedicated birds than in unvaccinated birds given narasin. The number of oocysts shed in the **vaccinated** groups was very low, but during a subsequent challenge with E. maxima and E. **tenella** the birds' immunity was found to be satisfactory.

Record Date Created: 19990913

11/7/36 (Item 6 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

10145945 99133822 PMID: 9950323

Immune responses in chickens against **Eimeria tenella** sporozoite antigen.

Garg R; Banerjee D P; Gupta S K

Department of Veterinary Parasitology, CCS Haryana Agricultural University, Hisar, India.

Veterinary parasitology (NETHERLANDS) Feb 1 1999, 81 (1) p1-10, ISSN 0304-4017 Journal Code: 7602745

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Two-day old broiler chicks were subcutaneously **immunized** with **Eimeria tenella** sporozoite antigen (25 microg per chick) with



or without adjuvants on 2 and 18 days of age and the effect of induced immunity was determined by challenging the chickens with 10(4) homologous sporulated oocysts at 32 days of age. Chicks **immunized** with sporozoite antigen emulsified in Freund's Complete Adjuvant (FCA) showed protection in terms of oocyst production, mortality and mean lesion scores. Antigen emulsified in FCA produced significant cell mediated immune responses (as assessed by lymphocyte migration inhibition test) from 12 to 30 days post-**immunization**. Antibody responses as assessed by enzyme linked immunosorbent assay were significant from 12 days postimmunization when the antigen was administered with or without adjuvants by subcutaneous route.

Record Date Created: 19990402

11/7/37 (Item 7 from file: 155)  
DIALOG(R) File 155:MEDLINE(P)

09982165 98430859 PMID: 9760059

Selection and development of a Spanish precocious strain of **Eimeria necatrix**.

Montes C; Rojo F; Hidalgo R; Ferre I; Badiola C

Merck Sharp and Dohme Research Laboratories, Madrid, Spain.

Veterinary parasitology (NETHERLANDS) Aug 14 1998, 78 (3) p169-83,  
ISSN 0304-4017 Journal Code: 7602745

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

A precocious line of **Eimeria necatrix** (PEN E-281/20) with an abbreviated life cycle was derived from a Spanish field strain (E-281) by repeated passages of the first shed oocysts recovered from the caecal contents of previously infected chickens. After 20 passages, the 'useful' prepatent period (time from infection to obtaining sufficient oocysts to repassage) of the parasite was reduced by 30 h (from 148 to 118 h). The earliest oocysts found in the caecal content were 114 h postinfection (hpi), on the 19th passage. The pathogenicity of the parasite was reduced in comparison with the parent strain, its immunogenicity against homologous and heterologous strains was maintained and its reproductive capacity was similar to or higher than that of the parent strain. Compared with the parent strain, the second generation of schizonts was reduced in size (reduced pathogenicity), third generation schizonts were bigger and with more merozoites (maintenance of the reproductive index) and the life cycle progressed faster from the second generation of schizonts (reduction of prepatent period). Complete second schizogony, from trophozoites to mature schizonts was observed frequently in the caeca of birds infected with both parent and precocious lines.

Record Date Created: 19981022

11/7/38 (Item 8 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

09440763 97331870 PMID: 9188177

Attempts to **immunize** chickens against *Cryptosporidium baileyi* with *C. parvum* oocysts and Paracox **vaccine**.

Sreter T; Hornok S; Varga I; Bekesi L; Szell Z

Department of Parasitology and Zoology, University of Veterinary Science, Budapest, Hungary.

Folia parasitologica (CZECH REPUBLIC) 1997, 44 (1) p77-80, ISSN 0015-5683 Journal Code: 0065750

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

To study the possibility of **immunization** against *Cryptosporidium*

baileyi Current, Upton et Haynes, 1986 with the attenuated anticoccidial **vaccine**, Paracox and oocysts of *C. parvum* Tyzzer, 1912, chickens were inoculated orally with either 3 x 10(3) **vaccine** oocysts or 8 x 10(5) *C. baileyi* or *C. parvum* oocysts at 1 week of age. The inoculation with Paracox **vaccine** and *C. parvum* oocysts was repeated at 2 and 3 weeks of age. Uninfected birds served as controls. All animals with the exception of one uninfected group were challenged orally with either 8 x 10(5) *C. baileyi* or 3 x 10(5) **Eimeria tenella** Railliet et Lucet, 1891 oocysts at 4 weeks of age. Sera were collected at 4 weeks of age, and were examined by ELISA using *C. baileyi* antigens. Birds inoculated with *C. parvum* oocysts did not shed *C. parvum* oocysts in their faeces, but anticryptosporidial antibodies could be detected in the sera. The total oocyst output of *C. parvum* inoculated chickens was 17% of that of previously uninfected birds after the oral challenge with *C. baileyi*. Considering that antibodies play no or only a minor role in resistance to *C. baileyi*, these results suggest that inoculation of chickens with *C. parvum* oocysts stimulated also cellular immune response. Based on the relative body weight gain, faecal scores, oocyst output, mortality, and caecal lesions in the birds **immunized** with Paracox **vaccine** and challenged with *E. tenella*, the **vaccination** induced only a moderate protection against the reinfection. The results of cross-**immunization** of chickens with *Eimeria* spp. and *C. baileyi* suggest that attenuated anti-eimerian **vaccines** do not induce any protection against cryptosporidial infection.

Record Date Created: 19970721

11/7/39 (Item 9 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

09103806 97009843 PMID: 8856947  
Immunisation of calves against **Eimeria alabamensis** coccidiosis.  
Svensson C; Olofsson H; Uggla A  
Swedish University of Agricultural Sciences, Skara, Sweden.  
Applied parasitology (GERMANY) Sep 1996, 37 (3) p209-16, ISSN  
0943-0938 Journal Code: 9308726  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed

Twelve calves which had been immunised with a trickle dose of altogether 100,000 oocysts of **Eimeria alabamensis** 16 days before turnout and 12 uninoculated calves were monitored during their first 20 days of grazing on a pasture naturally contaminated with oocysts of *E. alabamensis*. Eleven of the uninoculated calves developed gruel-like diarrhoea 3-6 days after turnout and excreted more than 850,000 oocysts/g of faeces (OPG) a few days later. In contrast, none of the immunised calves developed clinical coccidiosis and most of them excreted only a few oocysts. They lost on average 18 kg less in bodyweight than the unimmunised control calves. On day 21 all the calves were rehoused and on day 27 they were challenged with 10 million sporulated oocysts of *E. alabamensis* and turned out onto the same pasture. Only insignificant clinical signs were observed in 2 of the immunised calves and in one of the control calves. It was concluded that immunisation is a promising control measure for *E. alabamensis* coccidiosis. However, fewer or attenuated oocysts must be used, as 9 of the 12 inoculated calves developed clinical coccidiosis before turnout as a result of the immunisation doses.

Record Date Created: 19961113

11/7/40 (Item 10 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

08897662 96263519 PMID: 8654044  
Passive **immunization** against somatostatin increases resistance to

**Eimeria** vermiformis infection in susceptible mice.

Yun C H; Estrada A; Gajadhar A A; Redmond M J; Laarveld B  
Department of Animal and Poultry Science, University of Saskatchewan,  
Saskatoon, Canada.

Comparative immunology, microbiology and infectious diseases (ENGLAND)  
Jan 1996, 19 (1) p39-46, ISSN 0147-9571 Journal Code: 7808924

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The effect of in vivo immunoneutralization of somatostatin (SRIF) on **Eimeria** vermiformis intestinal infection was studied in resistant (BALB/c), and susceptible (C57BL/6) mouse strains. An anti-SRIF monoclonal antibody (Mab-SRIF) was used to passively immunize the mice by intraperitoneal injection. The animals were subsequently orally infected with oocysts of *E. vermiformis*. Individual fecal samples were collected daily for 21 days to monitor the kinetics of oocyst shedding. The fecal oocyst shedding was significantly higher in the C57BL/6 strain than in the BALB/c strain ( $P < 0.01$ ). Passive immunization with Mab-SRIF in the C57BL/6 mice significantly reduced the number of oocysts in feces ( $P < 0.05$ ), when compared to the infected non-immunized mice of the same strain. Infected BALB/c mice showed no difference in oocyst shedding in response to the passive immunoneutralization with Mab-SRIF. In conclusion, passive immunization with Mab-SRIF increased resistance to *E. vermiformis*-infection in the susceptible C57BL/6 mice, but not in the resistant BALB/c mice. This suggests that SRIF modulates gut immune function in parasitic infection.

Record Date Created: 19960801

11/7/41 (Item 11 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

08783192 96145985 PMID: 8560713

A live attenuated vaccine for the control of avian coccidiosis: trials in broiler breeders and replacement layer flocks in the United Kingdom.

Shirley M W; Bushell A C; Bushell J E; McDonald V; Roberts B

Institute for Animal Health, Compton Laboratory, Newbury.

Veterinary record (ENGLAND) Oct 28 1995, 137 (18) p453-7, ISSN 0042-4900 Journal Code: 0031164

Document type: Clinical Trial; Controlled Clinical Trial; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Losses caused by coccidiosis are a significant problem in the rearing of breeder and layer flocks. A live vaccine has been developed that contains attenuated lines of the seven species of **Eimeria** that infect the chicken. The attenuated lines were derived from virulent strains by selection for earlier development in chickens. In 11 field trials, the performance of vaccinated chicks was compared with that of matched controls receiving conventional drug prophylaxis. The vaccine was given in the drinking water to 116,600 young chickens and provided excellent control of coccidiosis. The occurrence of coccidial oocysts in the litter, coccidial lesions post mortem and overt coccidiosis was markedly lower in the vaccinated birds than in the controls.

Record Date Created: 19960228

11/7/42 (Item 12 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

05503090 87253655 PMID: 3598799

Transport of **Eimeria necatrix** sporozoites in the chicken: effects of irritants injected intraperitoneally.

Al-Attar M A; Fernando M A  
Journal of parasitology (UNITED STATES) Jun 1987, 73 (3) p494-502,  
ISSN 0022-3395 Journal Code: 7803124  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed

Light and electron microscopic observations confirmed that **Eimeria necatrix** sporozoites first enter villous epithelial cells of the chicken small intestine and are transported to the crypts by mononuclear cells. Ultrastructurally, these cells resemble granulated intraepithelial lymphocytes (IEL) rather than macrophages, as suggested previously. The **injection** of chickens intraperitoneally (i.p.) with a variety of irritants, including proteose peptone, at the time of oocyst inoculation or up to 12 hr postinoculation (PI) resulted in a delay in the arrival of sporozoites at the crypt. Significantly fewer sporozoites had arrived at the crypt by 24 hr PI in i.p.-**injected** birds as compared to controls. This delay in the arrival of sporozoites at the crypts was reflected by a delay in the development of intestinal lesions and in peak oocyst production. However, there was no significant decrease in the total numbers of oocysts produced by these birds as compared to controls, indicating that no significant loss of sporozoites occurs during the possible rerouting of the parasites. The presence of infective stages in extraintestinal sites was detected by transferring various tissues to coccidia-free recipients. Infection was transferable by gut, liver, and spleen from irritant-**injected** and control birds at all time intervals studied (12, 24, 36, and 48 hr PI). Infection was also transferable with blood and kidney, but not consistently. A small number of oocysts was passed by the recipients of peritoneal wash from irritant-**injected** birds at 12 hr PI. In all transfers, the prepatent period was normal, suggesting that the migrant stages are sporozoites.

Record Date Created: 19870826

11/7/43 (Item 13 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

04384526 84069417 PMID: 6646805

Studies to determine the taxonomic status of **Eimeria mitis**, Tyzzer 1929 and E. mivati, Edgar and Seibold 1964.

Shirley M W; Jeffers T K; Long P L

Parasitology (ENGLAND) Oct 1983, 87 (Pt 2) p185-98, ISSN 0031-1820  
Journal Code: 0401121

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

We have examined several taxonomic features of a number of strains of **Eimeria** from many sources world wide. The strains were isolated on the basis of their small spherical (or sub-spherical) oocysts. From a study of mean oocyst dimensions, electrophoretic variation of enzymes, ability to develop in embryonated **eggs**, absence of gross lesions in heavily infected chickens, and cross-immunity, all the strains were found to belong to one species. For convenience, the parasites when isolated, were referred to as strains of **E. mitis**/mivati-type, but after characterization they were clearly found to be **E. mitis**. In contrast, a laboratory strain of **E. mivati** supplied to one of us (M.W.S.) was found to be a mixture of **E. acervulina** and **E. mitis**. Evidence from these and other studies supports the notion that **E. mivati** is a nomina dubia.

Record Date Created: 19840107

11/7/44 (Item 14 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

03947275 82225323 PMID: 7201193

Demonstration of immunity against *Isospora suis* in swine.

Stuart B P; Sisk D B; Bedell D M; Gosser H S

Veterinary parasitology (NETHERLANDS) Feb 1982, 9 (3-4) p185-91,

ISSN 0304-4017 Journal Code: 7602745

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Piglets naturally exposed or experimentally infected with *Isospora suis* oocysts were given challenge doses of oocysts to determine the extent of development of immune resistance. Piglets in both studies shed low numbers of, or no detectable oocysts, following challenge. Administration of methylprednisolone acetate failed to induce oocyst shedding in previously infected piglets. Piglets rechallenged with *I. suis* following steroid **injections** also failed to shed significant numbers of oocysts suggesting development of immunity to reinfection.

Record Date Created: 19820807

11/7/45 (Item 15 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

03178557 79246004 PMID: 471536

A reappraisal of the taxonomic status of *Eimeria mivati* Edgar and Seibold 1964, by enzyme electrophoresis and cross-immunity tests.

Shirley M W

Parasitology (ENGLAND) Apr 1979, 78 (2) p221-37, ISSN 0031-1820

Journal Code: 0401121

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

An examination of 2 strains of *Eimeria acervulina* var. *mivati* (since 1973 *E. mivati* has been regarded as a variant of *E. acervulina*) showed that previous confusion over the taxonomic status of *E. mivati* arose because the investigations were done using laboratory cultures of *E. mivati* which were contaminated with *E. acervulina*. Electrophoretic analyses of enzymes, host specificity and cross-immunity tests have revealed that: (1) The 1971 Houghton strain of *E. acervulina* var. *mivati* was a mixture of 2 parasites. (a) Passage of this strain in embryonating **eggs** resulted in a selection against that parasite previously characterized as *E. acervulina*. (b) The parasite which did reproduce in **eggs** did not **immunize** chickens against subsequent challenge with *E. acervulina*. This parasite is most likely *E. mivati*. (c) *E. mivati* recovered from **eggs** did, however, **immunize** chickens against challenge with a new field strain which was morphologically identical to *E. mivati* and characterized by the same electrophoretic forms of 2 enzymes. (2) A strain of *E. acervulina* var. *mivati* from the USA was also a mixture of *E. acervulina* and *E. mivati*.

Record Date Created: 19791024

11/7/46 (Item 16 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

02062571 75144706 PMID: 1092227

Progress in the control of coccidiosis with anticoccidials and planned **immunization**.

Reid W M

American journal of veterinary research (UNITED STATES) Apr 1975, 36 (4 Pt 2) p593-6, ISSN 0002-9645 Journal Code: 0375011

Document type: Journal Article; Review

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

New anticoccidials continue to be introduced to the poultry industry on an average of about one every 2 years. Revised requirements have increased costs of discovery, development, and approval by the Food and Drug Administration to several million dollars for each product. Gross sales of anticoccidials are now estimated to be in excess of \$30,000,000 annually. Anticoccidials are almost universally used in starter rations for meat-type birds being raised under floor-pen management. Protection is more important with these fast-growing birds than with **egg**-producing types where immunity and cage layer management modify demands for anticoccidials. Coccidiosis mortality has generally been eliminated by all 25 approved anticoccidials if properly used. Selection of specific anticoccidials is based on the ability of the anticoccidial to: (1) improve weights and (2) feed conversion, and (3) to suppress development of lesions. Costs of the product may influence decisions on which one to use. With some anticoccidials the rapid emergence of drug resistant strains has been the biggest problem. "Switching" among the 13 classes of anticoccidials and the so-called "shuttle program" are common methods used to avoid development of resistance. Preventive medication will probably continue to constitute the major method of coccidiosis control with meat-type poultry if attempts to develop economic cage-type management do not supplant current litter and floor-pen management. Immunity acquired through incidental or planned **immunization** is more important in control of coccidiosis with layer and breeder flocks raised in floor-pens. Some progress in development of avirulent immunogenic strains of different species of coccidia has been reported and further research efforts in this direction should be encouraged. (26 Refs.)

Record Date Created: 19750702

11/7/47 (Item 17 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

01879278 74257835 PMID: 4209382

Immunity to **Eimeria** maxima: reactions of antisera in vitro and protection in vivo.

Rose M E

Journal of parasitology (UNITED STATES) Jun 1974, 60 (3) p528-30,  
ISSN 0022-3395 Journal Code: 7803124  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Record Date Created: 19740822

11/7/48 (Item 18 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

01785892 74084375 PMID: 4773337

Buquinolate and **immunization** to **Eimeria acervulina**.

Leathem W D

Poultry science (UNITED STATES) Jul 1973, 52 (4) p1468-72, ISSN  
0032-5791 Journal Code: 0401150  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Record Date Created: 19740321

11/7/49 (Item 19 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

01757167 74051481 PMID: 4761772  
The immunity arising from continuous low-level infection with  
**Eimeria tenella**.

Joyner L P; Norton C C  
Parasitology (ENGLAND) Dec 1973, 67 (3) p333-40, ISSN 0031-1820  
Journal Code: 0401121  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Record Date Created: 19740207

11/7/50 (Item 20 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

01367107 72106576 PMID: 5168239  
**Eimeria brunetti**: cross infections in chickens  
immunized to *E. maxima*.

Hein H  
Experimental parasitology (UNITED STATES) Jun 1971, 29 (3) p367-74,  
ISSN 0014-4894 Journal Code: 0370713  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Record Date Created: 19720419

11/7/51 (Item 21 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

01320497 72069054 PMID: 5129805  
Immunity to coccidiosis: protective effects of transferred serum and  
cells investigated in chick embryos infected with **Eimeria**  
**tenella**.

Rose M E; Long P L  
Parasitology (ENGLAND) Oct 1971, 63 (2) p299-313, ISSN 0031-1820  
Journal Code: 0401121  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
Record Date Created: 19720226

11/7/52 (Item 1 from file: 399)  
DIALOG(R)File 399:CA SEARCH(R)  
(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

124333053 CA: 124(25)333053g PATENT  
Antibacterial avian myelomonocytic growth factor for birds  
INVENTOR(AUTHOR): Johnston, Paul A.; Bland, Molly M.; Kemper, April E.;  
Liu, Hong; Tyczkowski, Julius K.; Harding, Timothy W.  
LOCATION: USA  
ASSIGNEE: Embrex, Inc.  
PATENT: PCT International ; WO 9606170 A1 DATE: 960229  
APPLICATION: WO 95US8663 (950712) \*US 292854 (940819) \*US 467724 (950606)  
PAGES: 60 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: C12N-015/27A;  
C12N-001/21B; C12N-001/19B; C07K-014/53B; A61K-038/19B; A61K-039/00;  
C12N-001/21J; C12R-001/19J; C12N-001/19K; C12R-001/84K  
DESIGNATED COUNTRIES: AM; AT; AU; BB; BG; BR; BY; CA; CH; CN; CZ; DE; DK;  
EE; ES; FI; GB; GE; HU; IS; JP; KE; KG; KP; KR; KZ; LK; LR; LT; LU; LV; MD;  
MG; MN; MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; TJ; TT; UA  
DESIGNATED REGIONAL: KE; MW; SD; SZ; UG; AT; BE; CH; DE; DK; ES; FR; GB;

GR; IE; IT; LU; MC; NL; PT; SE; BF; BJ; CF; CG; CI; CM; GA; GN; ML; MR; NE;  
SN; TD; TG

SECTION:

CA201005 Pharmacology

CA212XXX Nonmammalian Biochemistry

IDENTIFIERS: chicken avian myelomonocytic growth factor antibacterial

DESCRIPTORS:

Eimeria tenella... Salmonella typhimurium... Salmonella...

antibacterial avian myelomonocytic growth factor for birds to prevent  
infection by

Hemopoietins, myelomonocytic growth factors...

avian; in ovo administration of antibacterial avian myelomonocytic  
growth factor of chicken for birds to prevent bacterial infection

Antibiotics... Bird... Chicken... Duck... Egg... Goose... Pheasant... Quail

... Turkey... Vaccines...

in ovo administration of antibacterial avian myelomonocytic growth  
factor of chicken for birds to prevent bacterial infection

Escherichia coli... Pichia pastoris...

recombinant prep. of avian myelomonocytic growth factor of chicken in

11/7/53 (Item 2 from file: 399)

DIALOG(R) File 399:CA SEARCH(R)

(c) 2002 AMERICAN CHEMICAL SOCIETY. All rts. reserv.

121278829 CA: 121(23)278829a JOURNAL

Infection-induced protective antibodies to Eimeria maxima in hen and  
maternal transmission of the immunity to hatchling chick

AUTHOR(S): Smith, N. C.; Wallach, M.; Miller, C. M. D.; Braun, R.;  
Eckert, J.

LOCATION: Inst. fuer Parasitol., Univ. Zuerich, CH-8057, Zuerich, Switz.

JOURNAL: Infect. Immun. DATE: 1994 VOLUME: 62 NUMBER: 11 PAGES:

4811-17 CODEN: INFIBR ISSN: 0019-9567 LANGUAGE: English

SECTION:

CA215010 Immunochemistry

IDENTIFIERS: IgG Eimeria max hen chick transmission, coccidiosis vaccine

DESCRIPTORS:

Antibodies... Chicken... Egg yolk... Eimeria maxima... Immunoglobulins, G...

infection-induced protective antibodies to Eimeria maxima in hen and  
maternal transmission of the immunity through egg yolk to hatchling  
chick

Coccidiosis... Vaccines...

infection-induced protective antibodies to Eimeria maxima in hen and  
maternal transmission of the immunity through egg yolk to hatchling  
chick and for identificaion of anticoccidal vaccine candidates

? e au=dibner j a

Ref	Items	Index-term
E1	0	*AU=DIBNER J A
E2	79	AU=DIBNER J J
E3	1	AU=DIBNER J U
E4	16	AU=DIBNER J.J.
E5	29	AU=DIBNER JJ
E6	3	AU=DIBNER JULIA
E7	10	AU=DIBNER JULIA J
E8	2	AU=DIBNER L A
E9	5	AU=DIBNER LG
E10	1	AU=DIBNER M
E11	88	AU=DIBNER M D
E12	2	AU=DIBNER M P

Enter P or PAGE for more

? e au=dibner j j

Ref	Items	Index-term
-----	-------	------------



E1 79 \*AU=DIBNER J J  
 E2 1 AU=DIBNER J U  
 E3 16 AU=DIBNER J.J.  
 E4 29 AU=DIBNER JJ  
 E5 3 AU=DIBNER JULIA  
 E6 10 AU=DIBNER JULIA J  
 E7 2 AU=DIBNER L A  
 E8 5 AU=DIBNER LG  
 E9 1 AU=DIBNER M  
 E10 88 AU=DIBNER M D  
 E11 2 AU=DIBNER M P  
 E12 40 AU=DIBNER M.D.

Enter P or PAGE for more

? s e1 or e3 or e4 or e5 or e6

79 AU=DIBNER J J  
 16 AU=DIBNER J.J.  
 29 AU=DIBNER JJ  
 3 AU=DIBNER JULIA  
 10 AU=DIBNER JULIA J

S12 137 AU='DIBNER J J' OR AU='DIBNER J.J.' OR AU='DIBNER JJ' OR  
 AU='DIBNER JULIA' OR AU='DIBNER JULIA J'

? rd s12

...examined 50 records (50)  
 ...examined 50 records (100)  
 ...completed examining records  
 S13 63 RD S12 (unique items)

? t s13/7/all

>>>Format 7 is not valid in file 143

13/7/1 (Item 1 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

13634147 BIOSIS NO.: 200200262968  
 mRNA that encode for proteins capable of transporting L-methionine and/or  
 dl-2-hydroxy-4(methylthio) butanoic acid are present in the intestinal  
 epithelium of broilers.  
 AUTHOR: Pan Y-X(a); Wong E A(a); **Dibner J J**; Webb K E Jr(a)  
 AUTHOR ADDRESS: (a)Virginia Tech, Blacksburg, VA\*\*USA  
 JOURNAL: Journal of Dairy Science 84 (Supplement 1):p324 2001  
 MEDIUM: print  
 CONFERENCE/MEETING: Joint Meeting of the American Dairy Science  
 Association, American Meat Science Association, American Society of Animal  
 Science and the Poultry Science Association Indianapolis, Indiana, USA  
 July 24-28, 2001  
 ISSN: 0022-0302  
 RECORD TYPE: Citation  
 LANGUAGE: English

13/7/2 (Item 2 from file: 5)  
 DIALOG(R)File 5:Biosis Previews(R)  
 (c) 2002 BIOSIS. All rts. reserv.

13633979 BIOSIS NO.: 200200262800  
 Differential response of D- and L-Met free plasma in cows fed different  
 sources of rumen protected Met.  
 AUTHOR: Vazquez-Anon Mercedes(a); Parker David(a); **Dibner Julia**(a)  
 AUTHOR ADDRESS: (a)Novus International, Inc., Saint Louis, MO\*\*USA  
 JOURNAL: Journal of Dairy Science 84 (Supplement 1):p284 2001  
 MEDIUM: print  
 CONFERENCE/MEETING: Joint Meeting of the American Dairy Science  
 Association, American Meat Science Association, American Society of Animal  
 Science and the Poultry Science Association Indianapolis, Indiana, USA

July 24-28, 2001  
ISSN: 0022-0302  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/3 (Item 3 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

13605766 BIOSIS NO.: 200200234587  
Viability assay for sporocyst-forming protozoa.  
AUTHOR: **Dibner Julia**(a); Kitchell Marianne L; Pfannenstiel Mary Ann  
AUTHOR ADDRESS: (a)Chesterfield, MO\*\*USA  
JOURNAL: Official Gazette of the United States Patent and Trademark Office  
Patents 1255 (1):pNo Pagination Feb. 5, 2002  
MEDIUM: e-file  
ISSN: 0098-1133  
DOCUMENT TYPE: Patent  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: A method is disclosed for determining the viability of sporocyst-forming protozoa. The method involves treating a sample of protozoa with at least one vital dye and determining the viability of the protozoa in the sample by differential staining. The viability of protozoa in the sample can then be correlated with the viability of protozoa in the population from which the sample was obtained.

13/7/4 (Item 4 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

13603010 BIOSIS NO.: 200200231831  
Poly(A)+ RNA encoding proteins capable of transporting L-methionine and/or DL-2-hydroxy-4-(methylthio) butanoic acid are present in the intestinal mucosa of broilers.  
AUTHOR: Pan YuanXiang; Wong Eric A; **Dibner Julia J**; Vazquez-Anon Mercedes; Webb Kenneth E Jr(a)  
AUTHOR ADDRESS: (a)Department of Animal and Poultry Sciences, Virginia Polytechnic Institute and State University, Blacksburg, VA, 24061\*\*USA  
E-Mail: webbk@vt.edu  
JOURNAL: Journal of Nutrition 132 (3):p382-386 March, 2002  
MEDIUM: print  
ISSN: 0022-3166  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: To investigate the presence of poly(A)+ RNA that encode proteins capable of transporting L-methionine (L-Met) and/or DL-2-hydroxy-4-(methylthio) butanoic acid (HMB), *Xenopus* oocytes were injected with poly(A)+ RNA isolated from broiler intestinal mucosa. Healthy oocytes at stage V or VI were collected from *Xenopus laevis* and microinjected with water, poly(A)+ RNA or size-fractionated poly(A)+ RNA. The ability of the injected oocytes to take up either L-Met or HMB was examined by incubating oocytes with (methyl-3H)-L-Met or (5-14C)-HMB. A greater uptake of L-Met ( $P<0.01$ ) and HMB ( $P<0.05$ ) by oocytes injected with poly(A)+ RNA from the duodenum, jejunum and ileum of the small intestine was observed compared with water-injected oocytes. The greatest ( $P<0.05$ ) uptake occurred when poly(A)+ RNA from the jejunum or ileum was injected. Injections from four different pools of sucrose gradient-fractionated poly(A)+ RNA from all three intestinal segments induced ( $P<0.01$ ) L-Met uptake. There were three to four different pools

of sucrose gradient-fractionated poly(A)+ RNA from the duodenum, jejunum and ileum that induced ( $P < 0.05$ ) HMB uptake. Uptake of HMB was greater at pH 5.5 than at pH 7.5 and was independent of  $\text{Na}^+$ . Uptake of L-Met induced by all four poly(A)+ RNA pools decreased dramatically when  $\text{Na}^+$  was removed from the uptake buffer, which indicated that the majority of L-Met uptake was  $\text{Na}^+$ -dependent. These results indicate that there are multiple sized poly(A)+ RNA that encode proteins capable of mediated transport of L-Met and/or HMB present in broiler intestinal mucosa.

13/7/5 (Item 5 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

13484976 BIOSIS NO.: 200200113797  
Nutrient formulation and process for enhancing the health, livability, cumulative weight gain or feed efficiency in poultry and other animals.  
AUTHOR: Ivey Francis J; **Dibner Julia J**; Knight Christopher D  
JOURNAL: Official Gazette of the United States Patent and Trademark Office Patents 1253 (2):pNo Pagination Dec. 11, 2001  
MEDIUM: e-file  
ISSN: 0098-1133  
DOCUMENT TYPE: Patent  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: A nutrient formulation including moisture which is designed for use in poultry and other animals, and a method of feeding it which improves subsequent livability, cumulative feed efficiency and weight gain is disclosed.

13/7/6 (Item 6 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

13284424 BIOSIS NO.: 200100491573  
The hepatic extraction of plasma free amino acids and response to hepatic portal venous infusion of methionine sources in anesthetized SCWL males (*Gallus domesticus*).  
AUTHOR: Song Z; Beers K; **Dibner J J**; Vazquez-Anon M; McNew R; Bottje W(a)  
AUTHOR ADDRESS: (a)Department of Poultry Science, Center of Excellence for Poultry Science, University of Arkansas, Fayetteville, AR, 72701:  
wbottje@uark.edu\*\*USA  
JOURNAL: Comparative Biochemistry and Physiology Part B Biochemistry & Molecular Biology 130B (2):p237-250 September, 2001  
MEDIUM: print  
ISSN: 1096-4959  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English  
SUMMARY LANGUAGE: English

ABSTRACT: This study was conducted to investigate the hepatic extraction of plasma free amino acids in anesthetized Single Comb White Leghorn (SCWL) males (*Gallus domesticus*). SCWL males were anesthetized and implanted with cannulae in the carotid artery, hepatic vein, hepatic portal vein and the left hepatic duct. Free amino acids in plasma and bile were determined before, during and after 30-min infusions of Saline (control), DL-Methionine (DL-Met) or DL-2-hydroxy-4-methylthio-butanoic acid (DL-HMB) into the hepatic portal vein. Hepatic extraction rates (HER) of amino acids were calculated based on the concentration of amino acids in plasma multiplied by estimations of blood flow in the hepatic portal vein, hepatic artery and hepatic vein. For the non-essential amino acids,

alanine had the highest HER (46%). The liver also removed more than 20% of hepatic inflow of tyrosine and asparagine with substantial extraction (14-18%) of serine, glycine and glutamine, also. In contrast, less than 5% of hepatic inflow of glutamate and cystine were removed by liver. For the essential amino acids, HER for methionine, histidine and phenylalanine were 30, 14 and 17%, respectively, with less than 5% for branched-chain amino acids, lysine, arginine and threonine. Biliary secretion of amino acids represented a small percentage (<0.2%) of total hepatic extraction turnover of the amino acids. Infusion of methionine sources, DL-Met and DL-HMB, had no effect on hepatic metabolism of amino acids other than methionine. The results demonstrated for the first time, the hepatic extraction of circulating free amino acids in avian species in vivo.

13/7/7 (Item 7 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

12815172 BIOSIS NO.: 200100022321  
Absorption of 2-hydroxy-4-(methylthio)butanoic acid by isolated sheep  
ruminal and omasal epithelia.  
AUTHOR: McCollum M Q; Vazquez-Anon M; **Dibner J J**; Webb K E Jr(a)  
AUTHOR ADDRESS: (a)3020 Litton Reaves Hall, Blacksburg, VA, 24061-0306:  
webb-k@vt.edu\*\*USA  
JOURNAL: Journal of Animal Science 78 (4):p1078-1083 April, 2000  
MEDIUM: print  
ISSN: 0021-8812  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English  
SUMMARY LANGUAGE: English

ABSTRACT: Alimet (Novus Inter., Inc., St. Louis, MO) feed supplement (an 88% aqueous solution of 2-hydroxy-4-(methylthio) butanoic acid; HMB) is a source of L-Met commonly used in nonruminants and ruminants. The absorption of HMB across ovine omasal and ruminal epithelia was evaluated in this study. Ruminal and omasal epithelia were collected from eight lambs (BW = 67.6 kg  $\pm$  9.1) and mounted in parabolic chambers that were repeatedly sampled throughout a 60-min incubation. The appearance of HMB (using DL-(5-14C)-HMB as a radiolabeled marker) in serosal buffers increased quadratically ( $P < .004$ ) with time in both tissues. More ( $P < .001$ ) HMB appeared in the serosal buffers with omasal than with ruminal epithelia. Both tissues responded similarly, and, after 60 min of incubation, the accumulation of HMB within the tissues increased linearly ( $P < .001$ ) as substrate concentration (.375, .75, 1.5, 3.0, 6.0, and 12.0 mM) increased in mucosal buffers. As the concentration of HMB in the mucosal buffers increased, there was a quadratic ( $P < .001$ ) increase in the appearance of HMB in the serosal buffer of the omasal epithelium, indicating some saturation of the system. The increase in serosal appearance of HMB was linear ( $P < .001$ ) with ruminal tissue. The results indicate that there are probably multiple mechanisms involved in the absorption of HMB. Because saturation was observed in the omasum, it is likely that mediated transport accounts for at least a portion of the absorption of HMB in the omasum. Other mechanisms (e.g., diffusion and/or paracellular absorption) are responsible for the balance of the absorption. Omasal epithelium appears to have a greater capacity for HMB absorption than ruminal epithelium. The enzymes involved in the conversion of HMB to 2-keto-4-(methylthio)butanoic acid were found in ruminal and omasal epithelia, liver and kidney. These results indicate that HMB can be absorbed across ruminal and omasal epithelium and that HMB can be used as a source of L-methionine.

13/7/8 (Item 8 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

12522799 BIOSIS NO.: 200000276301

Nutrient formulation and process for feeding young poultry and other animals.

AUTHOR: Ivey Francis J(a); **Dibner Julia J**; Knight Christopher D

AUTHOR ADDRESS: (a)St. Louis, MO\*\*USA

JOURNAL: Official Gazette of the United States Patent and Trademark Office  
Patents 1228 (3):pNo pagination Nov. 16, 1999

MEDIUM: e-file.

ISSN: 0098-1133

DOCUMENT TYPE: Patent

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: A nutrient formulation including moisture which is designed for use in poultry and other animals, and a method of feeding it which improves subsequent livability, cumulative feed efficiency and weight gain is disclosed. The method comprises making available for consumption ad libitum a high moisture material containing at least about 20% by weight water to the poultry or other animals before they are offered dry food ad libitum.

13/7/9 (Item 9 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

12467459 BIOSIS NO.: 200000220961

Proper handling and fixation of gastrointestinal tissues of birds.

AUTHOR: Kitchell M L(a); **Dibner J J**

AUTHOR ADDRESS: (a)Novus International, 20 Research Park, St. Charles, MO,  
63304\*\*USA

JOURNAL: Journal of Histotechnology 23 (1):p57-60 March, 2000

ISSN: 0147-8885

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

SUMMARY LANGUAGE: English

ABSTRACT: Much attention is directed to the development and adaptation of the avian gastrointestinal system in order to evaluate feed additives and product candidates. Specific parameters going beyond cell population and morphology are repeatedly used for evaluation purposes. Measurements of the potential absorptive qualities of the intestine are compared, and good quality cross-sections of each specimen are critical. Good cross-sections with distinct and complete villi are dependent on fixation. Fixation variables include time, temperature, size of specimen, density of specimen, selected fixative, penetration, total volume of fixative, agitation of fixative and tissue, and the condition of the specimen when placed into fixative.

13/7/10 (Item 10 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

12280760 BIOSIS NO.: 200000034262

Intestinal villus growth, enterocyte migration and proliferation of the turkey poult.

AUTHOR: Applegate T J(a); **Dibner J J**; Kitchell M L; Uni Z; Lilburn M  
S(a)

AUTHOR ADDRESS: (a)Department of Animal Sciences, Ohio State  
University/O.A.R.D.C., Columbus, OH\*\*USA

JOURNAL: Poultry Science 78 (SUPPL. 1):p90 1999  
CONFERENCE/MEETING: Eighty-Eighth Annual Meeting of the Poultry Science Association, Inc. Springdale, Arkansas, USA August 8-11, 1999  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/11 (Item 11 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

11916288 BIOSIS NO.: 199900162397  
An alternate nutritional regime for starting broiler chicks.  
AUTHOR: Thaxton J P(a); **Dibner J J**  
AUTHOR ADDRESS: (a)Poultry Sci. Dep., Mississippi State Univ., Mississippi State, MS 39762\*\*USA  
JOURNAL: Poultry Science 77 (SUPPL. 1):p147 1998  
CONFERENCE/MEETING: Nineteenth Annual Meeting of the Southern Poultry Science Society and the 39th Annual Meeting of the Southern Conference on Avian Diseases Atlanta, Georgia, USA January 19-20, 1998  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/12 (Item 12 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

11808691 BIOSIS NO.: 199900054800  
Optimum dietary arginine:lysine ratio for broiler chickens is altered during heat stress in association with changes in intestinal uptake and dietary sodium chloride.  
AUTHOR: Brake J(a); Balnave D; **Dibner J J**  
AUTHOR ADDRESS: (a)Dep. Poult. ?Sci., Box 7608, NC State Univ., Raleigh, NC 27695-7608\*\*USA  
JOURNAL: British Poultry Science 39 (5):p639-647 Dec., 1998  
ISSN: 0007-1668  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: 1. The effects of varying the dietary arginine:lysine (Arg:Lys) ratio for broiler chickens at thermoneutral and high temperatures was studied in a series of 5 experiments which measured intestinal epithelial transport or evaluated growth and food efficiency with practical diets or diets supplemented with L-arginine free base. 2. The growth studies showed that increasing the Arg:Lys ratio at high temperatures produced consistent improvements in food conversion without any loss in growth. 3. Increasing dietary sodium chloride concentration reduced the Arg:Lys ratio necessary for optimum food conversion. 4. Food conversion responses were improved whether L-arginine free base was used as a dietary supplement in place of an inert filler or practical diets with differing ingredients were used to vary the Arg:Lys ratio. 5. In the presence of an equimolar concentration of lysine the uptake of arginine by the intestinal epithelium of heat-stressed broilers was reduced significantly compared with that of broilers at thermoneutral temperatures. 6. The results indicate that the ideal amino acid balance for broilers varies with ambient temperature.

13/7/13 (Item 13 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

11423850 BIOSIS NO.: 199800205182

The relative effectiveness of 2-hydroxy-4-(methylthio) butanoic acid and DL-methionine in young swine.

AUTHOR: Knighth C D(a); Atwell C A; Wuelling C W; Ivey F J; **Dibner J J**

AUTHOR ADDRESS: (a)Novus International Inc., St. Charles, MO 63304\*\*USA

JOURNAL: Journal of Animal Science 76 (3):p781-787 March, 1998

ISSN: 0021-8812

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: We compared the effectiveness of 2-hydroxy-4-(methylthio) butanoic acid (HMB) and DL-methionine (DLM) as sources of L-methionine activity in methionine-deficient primary cultures of pig liver cells and methionine-deficient early-weaned pigs. Viable hepatocytes were obtained from minced pig liver and maintained in a high density, differentiated, nonproliferation cell culture system. Culture medium was supplemented with HMB, DLM, or Lmethionine, and cells were pulse-dosed with L(14C(U))leucine for 24 h to determine the level of protein synthesis. Leucine incorporation per milligram of protein indicated a six-to eightfold increase in protein synthesis ( $P < .01$ ) with methionine levels between 5 and 10 MUM, regardless of source of methionine activity. Two 24-pen replicate methionine dose titrations were conducted with 95 early-weaned commercial crossbred pigs. The pelleted corn, dried whey, and porcine plasma basal diet contained 1.5% lysine, .23% methionine, and .48% cystine and was supplemented with 0, .05, or .10% methionine activity as DLM or HMB for 21 d. There was a 134, 104, and 61% increase ( $P < .01$ ) in cumulative ADG for each successive week on study with a 30 and 19% improvement in feed/gain ( $P < .01$ ) after 7 and 14 d. Performance responses due to source of methionine activity did not differ and slope ratio potency determinations (gain vs intake of methionine source) of HMB vs DLM indicated a 119, 111, and 95% relative activity for cumulative weekly performance. These results support the hypothesis that HMB and DLM provide equimolar levels of methionine activity in swine.

13/7/14 (Item 14 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

11028711 BIOSIS NO.: 199799649856

Effect of Santoquin and oxidized fat on liver and intestinal glutathione in broilers.

AUTHOR: Wang Sui-Ying(a); Bottje Walter(a); Maynard Philip(a); **Dibner Julia**; Shermer William

AUTHOR ADDRESS: (a)Dep. Poultry Science, Cent. Excellence Poultry Science, Univ. Arkansas, Fayetteville, AK 72701\*\*USA

JOURNAL: Poultry Science 76 (7):p961-967 1997

ISSN: 0032-5791

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Experiments were conducted to determine effects of Santoquin (ethoxyquin) and oxidized fat on liver and intestinal reduced (GSH) and oxidized (GSSG) glutathione, and pulmonary hypertension syndrome (PHS) mortality. Male broilers were randomly assigned in a 2 times 2 factorial consisting of 3.5% normal (NF) or oxidized (OxF) fat with or without ethoxyquin (E). Body weights and feed intake were monitored weekly, and tissues obtained at 3 and 7 wk for GSH and GSSG analysis. Compared to the NF group, NF/E gained more weight during the starter (0 to 3 wk), but not the grower (4 to 7 wk) period. Birds fed NF/E or NF exhibited greater feed efficiency in the starter period and greater gains during the starter and grower periods than birds fed OxF or OxF/E. No differences in PHS mortality between treatments were observed. Birds fed OxF exhibited

lower liver GSSG at 3 wk than the other groups, but there were no differences in liver GSH. Duodenal GSH was higher in birds fed OxF/E than in birds of NF group at 3 and 7 wk. Ileal GSH was higher at 3 wk in OxF/E birds than in OxF birds, but no differences were observed at 7 wk. All tissues exhibited higher GSH levels at 7 wk than at 3 wk. Birds fed ethoxyquin, regardless of fat source, exhibited higher duodenal GSH at 3 and 7 wk and higher ileal GSH at 3 wk than birds that did not receive ethoxyquin. Higher GSH would be beneficial by enhancing protection of intestinal cells to deleterious effects of toxins or other forms of oxidative stress.

13/7/15 (Item 15 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10690800 BIOSIS NO.: 199799311945  
Feeding of oxidized fats to broilers and swine: Effects on enterocyte turnover, hepatocyte proliferation and the gut associated lymphoid tissue.  
AUTHOR: **Dibner J J**(a); Atwell C A; Kitchell M L; Shermer W D; Ivey F J  
AUTHOR ADDRESS: (a)Novus Int. Inc., 20 Research Park Dr., Mo. Res. Park, St. Charles, MO 63304\*\*USA  
JOURNAL: Animal Feed Science and Technology 62 (1):p1-13 1996  
ISSN: 0377-8401  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: This study was designed to examine the cellular effects of feeding oxidized fats to broilers in the presence or absence of the antioxidant Santoquin. Although bird performance may be affected by the addition of rancid feed ingredients in the diet, little is known about the mechanism responsible for performance effects. In this study, gain, feed conversion and hematocrit were negatively affected by the feeding of oxidized fat (4 meq kg-1 diet). All these effects were ameliorated by the inclusion of ethoxyquin in the diet at 125 ppm. Results indicated that the gastrointestinal epithelium responded to oxidant stress with an increase in cell turnover. Hepatic cell proliferation increased. The gut associated immune system was also affected. Although plasma cell numbers were not affected based on histology, the concentration of immunoglobulin in the intestinal tissue appeared to be lower. These indicators of oxidative stress may be related to the poor performance seen in animals fed unstabilized diets containing oxidized ingredients.

13/7/16 (Item 16 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

10651297 BIOSIS NO.: 199699272442  
The relative effectiveness of 2-hydroxy-4-(methylthio) butanoic acid, (HMB) and DL-methionine (DLM) in young swine.  
AUTHOR: Knight C D; Atwell C A; Wuelling C W; Ivey F J; **Dibner J J**  
AUTHOR ADDRESS: Novus Int. Inc., St. Charles, MO\*\*USA  
JOURNAL: Journal of Animal Science 74 (SUPPL. 1):p53 1996  
CONFERENCE/MEETING: 88th Annual Meeting of the American Society of Animal Science, Midwestern Section and the American Dairy Science Association, Midwestern Branch Des Moines, Iowa, USA March 18-20, 1996  
ISSN: 0021-8812  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/17 (Item 17 from file: 5)



DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

09982812 BIOSIS NO.: 199598437730

Sodium bicarbonate improves broiler performance at high temperatures when added to diet with wide arginine:lysine ratio.

AUTHOR: Balnave D(a); Brake J; **Dibner J J**

AUTHOR ADDRESS: (a)Dep. Animal Sci., Univ. Sydney, Camden, NSW 2570\*\*  
Australia

JOURNAL: Poultry Science 74 (SUPPL. 1):p63 1995

CONFERENCE/MEETING: Eighty-fourth Annual Meeting of the Poultry Science Association, Inc. Edmonton, Alberta, Canada August 14-18, 1995

ISSN: 0032-5791

RECORD TYPE: Citation

LANGUAGE: English

13/7/18 (Item 18 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

09427079 BIOSIS NO.: 199497435449

Wide arginine:lysine ratio ameliorates effect of heat stress in broilers.

AUTHOR: Brake J(a); Balnave D; **Dibner J J**

AUTHOR ADDRESS: (a)Dep. Poultry Sci., N.C. State Univ., Raleigh, NC  
27695-7608\*\*USA

JOURNAL: Poultry Science 73 (SUPPL. 1):p74 1994

CONFERENCE/MEETING: Eighty-Third Annual Meeting of the Poultry Science Association, Inc. Starkville, Mississippi, USA August 7-12, 1994

ISSN: 0032-5791

RECORD TYPE: Citation

LANGUAGE: English

13/7/19 (Item 19 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

09426999 BIOSIS NO.: 199497435369

Feeding of oxidized fats to broilers: Poor performance is associated with changes in intestinal microflora and nutrient uptake.

AUTHOR: Atwell C A; Pierson E E M; Wuelling C W; Shermer W D; **Dibner J J**

AUTHOR ADDRESS: Novus Int. Inc., 20 Research Park Dr., Mo. Res. Park, St. Charles, Mo 63304\*\*USA

JOURNAL: Poultry Science 73 (SUPPL. 1):p47 1994

CONFERENCE/MEETING: Eighty-Third Annual Meeting of the Poultry Science Association, Inc. Starkville, Mississippi, USA August 7-12, 1994

ISSN: 0032-5791

RECORD TYPE: Citation

LANGUAGE: English

13/7/20 (Item 20 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

09426998 BIOSIS NO.: 199497435368

Feeding of oxidized fats to broilers: Poor performance is associated with changes cell turnover and the gut associated immune system.

AUTHOR: **Dibner J J**; Kitchell M L; Andrews J T; Wehmeyer M E; Ivey F J

AUTHOR ADDRESS: Novus Int. Inc., 20 Research Park Dr., Mo. Res. Park, St. Charles, MO 63304\*\*USA

JOURNAL: Poultry Science 73 (SUPPL. 1):p47 1994

CONFERENCE/MEETING: Eighty-Third Annual Meeting of the Poultry Science

Association, Inc. Starkville, Mississippi, USA August 7-12, 1994  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/21 (Item 21 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

09267319 BIOSIS NO.: 199497275689  
Effect of intermittent periods of high environmental temperature on broiler performance responses to sources of methionine activity.  
AUTHOR: Knight C D; Wuelling C W; Atwell C A; **Dibner J J**(a  
AUTHOR ADDRESS: (a)Novus Int. Inc., 20 Research Park Drive, Missouri Res.  
Park, St. Charles, MO 63304\*\*USA  
JOURNAL: Poultry Science 73 (5):p627-639 1994  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: The objective of these experiments was to study the effect of intermittent high temperature (IHT) on performance of broilers fed different sources of methionine activity. Two 20-d experiments were conducted in which individually caged 28-d-old cockerels were exposed to a 5-d period of constant high temperature (HT, 30 to 32 C) followed by an equal period of thermoneutral (TN) temperature (22 C) with the 10-d temperature cycle repeated twice. Birds held at 22 C were TN controls. Grower diets contained 3,275 kcal/kg ME and 20% CP and were supplemented with either 2-hydroxy-4-(methylthio)butanoic acid (HMB, Alimet Feed Supplement) or DL-Met up to a maximum of .88 to .90% total sulfur amino acids (TSAA). In Experiment 1, gain:feed but not average daily gain was greater (P lt .05) for HMB than DL-Met birds subjected to IHT (.451 vs .413, respectively), but no treatment differences were observed for TN birds. Results of Experiment 2 demonstrated a linear response to HMB and DL-Met dose in TN (P lt .01); however, only HMB-supplemented birds responded similarly in IHT (P lt .01). These results are consistent with lower availability of DL-Met as a result of IHT. In vitro experiments indicated that capacity for uptake of DL-Met into intestinal epithelial cells was reduced in heat-stressed birds. Uptake of D-Met was more severely affected than was L-Met. Consequently, a third performance experiment compared the ability of D-Met and L-Met to support growth under conditions of HT. The results indicated that the effect of HT on broiler performance was mediated through reduced utilization of D-Met.

13/7/22 (Item 22 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

09138520 BIOSIS NO.: 199497146890  
Response of laying hens to diets containing up to 2 percent DL-methionine or equimolar (2.25 percent) 2-hydroxy-4-(methylthio)butanoic acid.  
AUTHOR: Wideman Robert F(a); Ford Bonnie C; **Dibner Julia J**; Robey W  
Wade; Yersin Andrew G  
AUTHOR ADDRESS: (a)Dep. Poultry Science, Univ. Arkansas, Fayetteville, AR  
72701\*\*USA  
JOURNAL: Poultry Science 73 (2):p259-267 1994  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Diets supplemented with up to .6% DL-Met (DLM) or .68%

2-hydroxy-4-(methylthio)butanoic acid (HMB, Alimet) acidify the urine and reduce the incidence of urolithiasis in pullets and laying hens. Excessive acidification potentially may reduce eggshell quality and bone mineralization by interfering with Ca metabolism and may severely challenge the liver and kidneys, which are the primary organs responsible for attenuating metabolic acidosis. To evaluate these possibilities, 30-wk-old Single Comb White Leghorn hens in full production (five hens per replicate, six replicates per diet treatment) were fed for 30 d a 15.7% CP corn and soybean meal-based control layer ration alone or supplemented with DLM (.5, 1, 1.5 or 2%) or equimolar HMB (.56, 1.13, 1.69, or 2.25%). None of the diets caused mortality or, gross hepatic or renal damage. Hens fed diets supplemented with the highest levels of DLM and HMB exhibited significant reductions in feed intake, hen-day egg production, and liver mass and had lower plasma concentrations of alanine amino-transferase and isocitrate dehydrogenase when compared with hens fed the control diet. Kidney mass was not significantly affected by high levels of DLM or HMB, but plasma uric acid was significantly higher in hens fed 2% DLM compared with hens fed the control diet. The highest levels of DLM and HMB did not significantly alter total plasma Ca or inorganic phosphate concentrations, nor were percentage eggshell or femur mineralization (femur ash mass: defatted bone mass, femur ash mass: bone volume) significantly reduced. These results indicate that very high dietary levels of both DLM (2%) and HMB (2.25%) cause feed avoidance and a corresponding reduction in egg production. Cessation of egg production probably accounted for the decrease in liver mass and for reduced plasma concentrations of hepatic enzymes. Feed avoidance may ameliorate the potential impact of very high levels of DLM and HMB on Ca metabolism and hepato-renal function.

13/7/23 (Item 23 from file: 5)  
DIALOG(R) File 5: Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08894274 BIOSIS NO.: 199396045775  
Immunohistochemical and nucleic acid analysis of somatotropin receptor populations in the bovine ovary.  
AUTHOR: Lucy Matthew C(a); Collier Robert J; Kitchell Marianne L; **Dibner Julia J**; Hauser Scott D; Krivi Gwen G  
AUTHOR ADDRESS: (a) Monsanto Co., AA3C, 700 Chesterfield Parkway North, St. Louis, MO 63198\*\*USA  
JOURNAL: Biology of Reproduction 48 (6):p1219-1227 1993  
ISSN: 0006-3363  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: Ovaries were analyzed for somatotropin receptor protein and mRNA through use of immunohistochemistry, solution hybridization/nuclease protection, Northern blotting, and reverse transcriptase polymerase chain reaction (RT-PCR). As indicated by immunoperoxidase staining, CL expressed immunoreactive somatotropin receptor (positive stain). Ovarian stroma, connective tissue, endothelium, and erythrocytes did not express somatotropin receptor (negative stain). Within the CL, somatotropin receptor protein was expressed primarily in large luteal cells whereas small luteal cells were negative. Most follicles (1-5 mm, after fixation) were negative for somatotropin receptor. On the basis of solution hybridization/nuclease protection, the mRNA for somatotropin receptor was found in greatest abundance in CL and large luteal cells and was nearly undetectable in small luteal cells or follicles (class 1, 3-5 mm; class 2, 6-9 mm; and class 3, greater than or equal to 10 mm). Northern blotting of mRNA for somatotropin receptor showed expression of somatotropin receptor mRNA transcripts in whole ovary (4.7 and 4.4 kb), CL (4.7 and 4.4 kb), and liver (4.4 kb); and RT-PCR amplified a single amino acid coding region for somatotropin receptor in CL and liver. In summary, somatotropin

receptor (both immunoreactive protein and mRNA) is found primarily in the large luteal cell, and lesser amounts of the expressed receptor or its message are found in the follicle. Alternative sizes of mRNA for somatotropin receptor suggest novel mRNA processing in the bovine ovary.

13/7/24 (Item 24 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08720121 BIOSIS NO.: 199395009472  
Effect of heat stress on 2-hydroxy-4-(methylthio)butanoic acid and DL-methionine absorption measured in vitro.  
AUTHOR: **Dibner J J**(a); Atwell C A; Ivey F J  
AUTHOR ADDRESS: (a)Julia Dibner Mail Zone BB21, Novus International, 700 Chesterfield Village Parkway, Chesterfield  
JOURNAL: Poultry Science 71 (11):p1900-1910 1992  
ISSN: 0032-5791  
DOCUMENT TYPE: Article  
RECORD TYPE: Abstract  
LANGUAGE: English

ABSTRACT: The objective of the present experiments was to determine the biochemical basis for preliminary chick performance data, which indicate an ameliorative effect of 2-hydroxy-4-(methylthio)butanoic acid (HMB) when compared with DL-methionine (DLM) fed during hot conditions. In vitro passage of HMB or DLM across intact segments of small intestine from either control (thermoneutral, TN) or heat-stressed (HS) birds was used as a model for intestinal absorption. For DLM placed in the lumen, appearance in the outside buffer was reduced when using intestine from HS birds compared with tissue from TN birds. In contrast, the appearance of HMB in the outside buffer was greater using HS intestine, resulting in a substrate by environment interaction (P lt .01). Slices of everted small intestine from TN and HS birds were used to study epithelial uptake of methyl labeled 14C-DLM by three transport pathways: diffusion, carrier-specific energy- and sodium-independent uptake (ESI), and carrier-specific energy- and sodium-dependent uptake (ESD). Correcting for extracellular volume, total epithelial uptake of 14C-DLM (diffusion plus ESI plus ESD) was reduced by 34% in HS intestine (P lt .05). Energy-dependent uptake was observed to decrease by 87% in HS (P lt .05). Energy-independent uptake was increased (136%, HS versus TN, P lt .05), but not enough to compensate for the decrease in ESD uptake. Intestinal transport systems for glucose and leucine were also observed to change during HS, suggesting a role for cellular transport changes in the performance reduction associated with HS.

13/7/25 (Item 25 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08674062 BIOSIS NO.: 199345092137  
Responses of laying hens to diets containing up to 2 percent DL-methionine (DLM) or equimolar (2.29 percent) levels of liquid methionine hydroxy analog free acid (MHA).  
AUTHOR: Wideman Robert F(a); Ford Bonnie C(a); **Dibner Julia J**; Robey W Wade; Yersin Andrew G  
AUTHOR ADDRESS: (a)Penn State Univ., University Park, PA 16802\*\*USA  
JOURNAL: Poultry Science 72 (SUPPL. 1):p70 1993  
CONFERENCE/MEETING: Eighty-second Annual Meeting of the Poultry Science Association, Inc. East Lansing, Michigan, USA July 26-29, 1993  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/26 (Item 26 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08674013 BIOSIS NO.: 199345092088  
Comparison of liver morphology in a commercial flock with liver spleen and kidney pathology after feeding high levels of DL-methionine or 2-hydroxy-4-(methylthio)butanoic acid (HMB, Alimet) to 30-week-old laying hens.  
AUTHOR: **Dibner J J**(a); Ivey F J(a); Kitchell M L(a); Robey W W(a); Yersin A G(a); Wideman R F  
AUTHOR ADDRESS: (a)Novus Int., St. Louis, MO 63141\*\*USA  
JOURNAL: Poultry Science 72 (SUPPL. 1):p54 1993  
CONFERENCE/MEETING: Eighty-second Annual Meeting of the Poultry Science Association, Inc. East Lansing, Michigan, USA July 26-29, 1993  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/27 (Item 27 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08673958 BIOSIS NO.: 199345092033  
Changes in intestinal structure and enteroendocrine function are associated with changes in nutrient uptake during acute heat stress.  
AUTHOR: Atwell C A; Kitchell M L; **Dibner J J**  
AUTHOR ADDRESS: Novus Int. Inc., Maryville Centre Drive, St. Louis, MO\*\* USA  
JOURNAL: Poultry Science 72 (SUPPL. 1):p35 1993  
CONFERENCE/MEETING: Eighty-second Annual Meeting of the Poultry Science Association, Inc. East Lansing, Michigan, USA July 26-29, 1993  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/28 (Item 28 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08664278 BIOSIS NO.: 199345082353  
Exposure of broilers to intermittent high temperature results in different performance responses to sources of methionine activity.  
AUTHOR: Knight C D; Wuelling C W; **Dibner J J**; Ivey F J  
AUTHOR ADDRESS: Novus Int. Inc., 530 Maryville Centre Dr., St. Louis, MO 63141\*\*USA  
JOURNAL: Poultry Science 72 (SUPPL. 1):p175 1993  
CONFERENCE/MEETING: Fourteenth Annual Meeting of the Southern Poultry Science Society Atlanta, Georgia, USA January 20-22, 1993  
ISSN: 0032-5791  
RECORD TYPE: Citation  
LANGUAGE: English

13/7/29 (Item 29 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08345566 BIOSIS NO.: 000043077689  
REDUCTION IN INTESTINAL UPTAKE OF DL METHIONINE DURING ACUTE HEAT STRESS IS ASSOCIATION WITH CHANGES IN EPITHELIAL TRANSPORT SYSTEMS  
AUTHOR: **DIBNER J J**; ATWELL C A; ANDREWS J T; KITCHELL M L; WILLIAMS D

C; WEYMEYER M E; WUELLING C W; IVEY F J  
AUTHOR ADDRESS: NOVUS INTERNATIONAL INC., 530 MARYVILLE CENTRE DR., ST.  
LOUIS, MO. 63141.  
JOURNAL: EIGHTY-FIRST ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION,  
FAYETTEVILLE, ARKANSAS, USA, AUGUST 3-6, 1992. POULT SCI 71 (SUPPL. 1).  
1992. 111. 1992  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/30 (Item 30 from file: 5)  
DIALOG(R)File 5:Biosis Previews(F)  
(c) 2002 BIOSIS. All rts. reserv.

08345460 BIOSIS NO.: 000043077583  
USE OF A MONOCLONAL ANTIBODY TO BROMODEOXYURIDINE TO STUDY THE  
PROLIFERATIVE SUBPOPULATION OF THE AVIAN GASTROINTESTINAL SYSTEM  
AUTHOR: **DIBNER J J**; KITCHELL M J  
AUTHOR ADDRESS: NOVUS INTERNATIONAL, MARYVILLE CENTRE DRIVE, ST. LOUIS, MO.  
63141.  
JOURNAL: EIGHTY-FIRST ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION,  
FAYETTEVILLE, ARKANSAS, USA, AUGUST 3-6, 1992. POULT SCI 71 (SUPPL. 1).  
1992. 75. 1992  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/31 (Item 31 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08345119 BIOSIS NO.: 000043077242  
METABOLIC DIFFERENCES BETWEEN 2 HYDROXY-4-METHYLTHIO BUTANOIC ACID HMB  
ALIMET AND DL METHIONINE DLM DURING HEAT STRESS  
AUTHOR: **DIBNER J J**; SWICK R A; KNIGHT C D; ATWELL C A; KITCHELL M L;  
IVEY F J  
AUTHOR ADDRESS: NOVUS INTERNATIONAL, 530 MARYVILLE CENTRE DRIVE, ST. LOUIS,  
MO. 63141.  
JOURNAL: THIRTEENTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY,  
ATLANTA, GEORGIA, USA, JANUARY 20-21, 1992. POULT SCI 71 (SUPPL. 1). 1992.  
147. 1992  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/32 (Item 32 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

08186334 BIOSIS NO.: 000094010107  
CAPACITY IN THE LIVER OF THE BROILER CHICK FOR CONVERSION OF SUPPLEMENTAL  
METHIONINE ACTIVITY TO L METHIONINE  
AUTHOR: **DIBNER J J**; IVEY F J  
AUTHOR ADDRESS: NOVUS INTERNATIONAL, MAIL ZONE BB21, 700 CHESTERFIELD  
PARKWAY NORTH, CHESTERFIELD, MO. 63198.  
JOURNAL: POULT SCI 71 (4). 1992. 700-708. 1992  
FULL JOURNAL NAME: Poultry Science  
CODEN: POSCA  
RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: The objective of the present experiments was to determine whether the levels of supplemental methionine sources currently used in practical diets exceed the capacity of the chick to convert the supplement to L-methionine. Supplemental sources examined included DL-2-hydroxy-4-(methylthio)butanoic acid (DL-HMB, Alimet, or MHA) and DL-methionine (DL-Met). Two approaches were taken: first, the amount of enzyme activity available for conversion of the two supplemental methionine sources was determined using optimum reaction conditions for each and chick liver homogenate as the enzyme source. These experiments showed that total liver enzyme conversion activity was 564  $\mu\text{mol/h}$  for DL-HMB and 529  $\mu\text{mol/h}$  for DL-Met. The total activities for the two sources were not different when measured at saturating substrate concentration. Second, to address the question of whether the enzyme is limiting for either source under practical feeding conditions, birds were fed starter diets supplemented with DL-HMB or DL-Met at .25% of the diet for 3 wk. When hepatic levels of free HMB and methionine were determined, birds fed DL-HMB contained 7.6 nmol HMB/g of liver and 84.7 nmol methionine/g of liver. Birds fed DL-Met had levels of 7.6 nmol HMB/g liver and 80.3 nmol methionine/g liver. These results indicate no accumulation of HMB or DL-Met, which occur if conversion capacity were saturated. By calculation, a bird consuming 100 g/day of a diet supplemented with .25% activity would need to convert about 70  $\mu\text{mol/h}$ , indicating a sevenfold excess of enzyme in the liver alone.

13/7/33 (Item 33 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07790424 BIOSIS NO.: 000041076375  
EFFECT OF METHIONINE SOURCE AND ELEVATED TEMPERATURE ON NITROGEN BALANCE IN BROILERS  
AUTHOR: SWICK R A; IVEY F J; **DIBNER J J**  
AUTHOR ADDRESS: ANIMAL SCI. DIV., MONSANTO CO., CHESTERFIELD, MO. 63198.  
JOURNAL: TWELFTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY, ATLANTA, GEORGIA, USA, JANUARY 28-29, 1991. POULT SCI 70 (SUPPL. 1). 1991. 185.  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/34 (Item 34 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07790341 BIOSIS NO.: 000041076292  
RELATIONSHIP OF PRACTICAL SUPPLEMENTAL LEVELS OF METHIONINE ACTIVITY TO CHICK LIVER ENZYME CONVERSION CAPACITY  
AUTHOR: **DIBNER J J**; IVEY F J  
AUTHOR ADDRESS: MONSANTO CO., CHESTERFIELD, MO.  
JOURNAL: TWELFTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY, ATLANTA, GEORGIA, USA, JANUARY 28-29, 1991. POULT SCI 70 (SUPPL. 1). 1991. 157.  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/35 (Item 35 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

07347689 BIOSIS NO.: 000090127591  
HEPATIC PROTEIN AND AMINO ACID METABOLISM IN POULTRY  
AUTHOR: **DIBNER J J**; IVEY F J  
AUTHOR ADDRESS: MONSANTO CO., ANIMAL SCI. DIV., 700 CHESTERFIELD VILLAGE  
PARKWAY, CHESTERFIELD, MO. 63198.  
JOURNAL: POULT SCI 69 (7). 1990. 1188-1194. 1990  
FULL JOURNAL NAME: Poultry Science  
CODEN: POSCA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: Two subjects are discussed: first, the regulation of hepatic protein synthesis; and second, the intermediary metabolism of methionine, particularly with respect to the role of 2-hydroxy-4-(methylthio)-butanoic acid (HMB, Alimet). In the first section, the regulation of albumin synthesis is reviewed in terms of molecular events associated with the changes in albumin synthesis during fasting and refeeding. The effect of infection or of inflammatory stress on both albumin and total protein synthesis in the liver is also discussed. In the second part, research results are presented which indicate that HMB is a naturally occurring compound in methionine intermediary metabolism. The HMB synthesis by chick liver enzymes is demonstrated, and its role in normal avian methionine metabolism is discussed.

13/7/36 (Item 36 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07275650 BIOSIS NO.: 000090055535  
2 HYDROXY-4-METHYLTHIOBUTANOIC ACID IS A NATURALLY OCCURRING METHIONINE  
PRECURSOR IN THE CHICK  
AUTHOR: **DIBNER J J**; DURLEY R C; KOSTEL J G; IVEY F J  
AUTHOR ADDRESS: MONSANTO COMPANY, ANIMALS SCI. DIVISION, CHESTERFIELD, MO.  
63198.  
JOURNAL: J NUTR 120 (6). 1990. 553-560. 1990  
FULL JOURNAL NAME: Journal of Nutrition  
CODEN: JONUA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The objective of these experiments was to determine the origin of 2-hydroxy-4-(methylthio)-butanoic acid (HMB) detected in the liver and excreta of chicks that had never been fed Alimet (an 88% aqueous solution of HMB) or MHA (an 86% calcium salt of HMB). Gas chromatography and mass spectrometry were used to identify HMB in these birds. A normal biochemical pathway of 5'-deoxy-5'-methylthioadenosine (MTA) is proposed as the source of naturally occurring HMB. Studies of conversion of [methyl-14C]MTA to L-methionine by chick liver enzymes showed that radiolabeled HMB and 2-oxo-4-(methylthio)butanoic acid (keto-methionine) were synthesized during the reaction. Specific radioactivities of labeled HMB and keto-methionine showed that HMB is not synthesized from keto-methionine. Fractionation studies indicated that radiolabeled HMB formed from [14C]MTA could be used in the synthesis of L-methionine in the presence of peroxisomes and/or mitochondrial enzymes. In this way, HMB synthesized from MTA by chick liver enzymes functions as a precursor of L-methionine and as an intermediate in a naturally occurring pathway for the synthesis of L-methionine in the chick.

13/7/37 (Item 37 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)



(c) 2002 BIOSIS. All rts. reserv.

07129058 BIOSIS NO.: 000039065752  
IMPACT OF METHIONINE SOURCES ON PERFORMANCE OF BROILERS GROWING UNDER WARM  
AND HUMID CONDITIONS  
AUTHOR: SWICK R A; CRESWELL D C; **DIBNER J J**; IVEY F J  
AUTHOR ADDRESS: ANIM. SCI. DIV., MONSANTO CO., CHESTERFIELD, MO. 63198,  
USA.  
JOURNAL: ELEVENTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY.  
POULT SCI 69 (SUPPL. 1). 1990. 194. 1990  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/38 (Item 38 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07128967 BIOSIS NO.: 000039065661  
2 HYDROXY-4-METHYLTHIOBUTANOIC ACID HMB ALIMET IS A NATURALLY OCCURRING  
METHIONINE SOURCE  
AUTHOR: **DIBNER J J**; IVEY F J  
AUTHOR ADDRESS: ANIM. SCI. DIV., MONSANTO CO., ST. LOUIS, MO.  
JOURNAL: ELEVENTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY.  
POULT SCI 69 (SUPPL. 1). 1990. 164. 1990  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/39 (Item 39 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

07118239 BIOSIS NO.: 000039054933  
THE EFFECT OF SULFUR AMINO ACID BALANCE ON THE PERFORMANCE OF 2  
HYDROXY-4-METHYLTHIOBUTANOIC ACID HMB ALIMET IN THE CRYSTALLINE AMINO  
ACID DIET  
AUTHOR: **DIBNER J J**; KNIGHT C D; IVEY F J  
AUTHOR ADDRESS: ANIMAL SCI. DIV., MONSANTO CO., CHESTERFIELD, MO.  
JOURNAL: 79TH ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION, INC.,  
BLACKSBURG, VIRGINIA, USA, MAY 1990. POULT SCI 69 (SUPPL. 1). 1990. 43.  
1990  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/40 (Item 40 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

06749785 BIOSIS NO.: 000088059216  
RAPID DETECTION OF PROLIFERATING CELLS USING MICROWAVE FIXATION AND A  
MONOCLONAL ANTIBODY TO BROMODEOXYURIDINE  
AUTHOR: KITCHELL M L; **DIBNER J J**  
AUTHOR ADDRESS: MONSANTO AGRIC. CO., ANIM. SCI. DIV., 700 CHESTERFIELD  
VILLAGE PARKWAY, CHESTERFIELD, MO. 63198.  
JOURNAL: J HISTOTECHNOL 12 (2). 1989. 101-103. 1989  
FULL JOURNAL NAME: Journal of Histotechnology  
CODEN: JOHID

RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The objective of this research was to develop a rapid morphological test for proliferating cells using diaminobenzidine as the substrate marker. This technique can be employed to visualize proliferating cells by using a monoclonal antibody to bromodeoxyuridine and a standard immunoperoxidase technique. This paper describes methods for the rapid demonstration of S-phase nuclei in routine paraffin tissue section. The tissue was fixed by exposure to microwave irradiation for various times and temperatures. The subsequent staining results are reliable and can easily fit into an existing immunoperoxidase program.

13/7/41 (Item 41 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

06505653 BIOSIS NO.: 000037077669  
2 HYDROXY-4-METHYLTHIOBUTANOIC ACID HMB ALIMET IS A NORMAL METABOLITE IN AVIAN METHIONINE METABOLISM  
AUTHOR: **DIBNER J J**; SWICK R A; KOSTELC J G; IVEY F J  
AUTHOR ADDRESS: ANIM. SCI. DIV., MONSANTO CO., CHESTERFIELD, MO. 63198.  
JOURNAL: TENTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY.  
POULT SCI 68 (SUPPL. 1). 1989. 179. 1989  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/42 (Item 42 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

06284318 BIOSIS NO.: 000086118501  
ABSORPTION OF CARBON-14 2 HYDROXY-4-METHYLTHIOBUTANOIC ACID ALIMET FROM THE HINDGUT OF THE BROILER CHICK  
AUTHOR: **DIBNER J J**; KNIGHT C D; SWICK R A; IVEY F J  
AUTHOR ADDRESS: MONSANTO COMPANY, ANIMAL SCI. DIV., ST. LOUIS, MO. 63198.  
JOURNAL: POULT SCI 67 (9). 1988. 1314-1321. 1988  
FULL JOURNAL NAME: Poultry Science  
CODEN: POSCA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The purpose of these studies was to examine the role of the hindgut of the broiler chick in the absorption of 2-hydroxy-4-(methylthio)butanoic acid (HMB). When <sup>14</sup>C-HMB was delivered directly into the hindgut, the rate of absorption from this gastrointestinal site was about 40% of the administered dose per hour. Plasma radiolabel appearance indicated that the <sup>14</sup>C-HMB lost from the hindgut was being absorbed into the bloodstream of the bird. Decarboxylation experiments using cecal microorganisms showed that the loss of <sup>14</sup>C-HMB could not be accounted for by bacterial metabolism. When birds were dosed with radiolabelled HMB and tissues samples were tested, results showed that the <sup>14</sup>C-HMB that was absorbed from the hindgut was incorporated into protein in a dose-related manner. In addition, and equimolar, equal specific activity intraperitoneal dose of HMB did not alter the rate of HMB absorption from the hindgut. This indicates that HMB absorption from the gut is not limited by HMB already in the body tissues. This result confirmed that the rate of HMB diffusion into the blood and its conversion to methionine in body tissues were sufficient to maintain the concentration gradient required for the continued absorption of HMB. Finally, whole body autoradiography comparing <sup>35</sup>S-HMB and

35S-DL-methionine showed no substantial differences in terms of label density or distribution. These demonstrate that 14C-HMB disappears from the lumen of the large intestine and ceca when it is administered directly into the hindgut. This research confirms that HMB is absorbed throughout the entire gastrointestinal system.

13/7/43 (Item 43 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

06003017 BIOSIS NO.: 000035094380  
COMPARISON OF ALIMET 2 HYDROXY-4-METHYLTHIOBUTANOIC ACID HMB AND DL METHIONINE DLM URINARY AND FECAL EXCRETION  
AUTHOR: **DIBNER J J**; SWICK R A; ONTIVEROS R R; EHMLER T J; KOSTELC J G  
; IVEY F J  
AUTHOR ADDRESS: ANIM. SCI. DIV., MONSANTO COMPANY, ST. LOUIS.  
JOURNAL: 77TH ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION, INC. POULT SCI 67 (SUPPL. 1). 1988. 76. 1988  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/44 (Item 44 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

05993448 BIOSIS NO.: 000035084811  
EXCRETION OF RADIOLABELLED METABOLITES FOLLOWING A DOSE OF CARBON-14 ALIMET 2 HYDROXY-4-METHYLTHIOBUTANOIC ACID HMB OR CARBON-14 DL METHIONINE DLM  
AUTHOR: **DIBNER J J**; KNIGHT C D; SWICK R A; IVEY F J  
AUTHOR ADDRESS: MONSANTO CO., ST. LOUIS, MO. 63198.  
JOURNAL: NINTH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY. POULT SCI 67 (SUPPL. 1). 1988. 12. 1988  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/45 (Item 45 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

05029191 BIOSIS NO.: 000031104323  
THE IMPACT OF DIFFERENT SOURCES OF METHIONINE ACTIVITY ON CHICK SKELETAL MUSCLE PROTEIN DEGRADATION MEASURED IN-VITRO  
AUTHOR: **DIBNER J J**; KNIGHT C D  
AUTHOR ADDRESS: MONSANTO COMPANY, ST. LOUIS, MISSOURI 63198.  
JOURNAL: 75TH ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION, INC. POULT SCI 65 (SUPPL. 1). 1986. 34. 1986  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/46 (Item 46 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04639008 BIOSIS NO.: 000079052045  
COMPARATIVE ABSORPTION OF 2 HYDROXY-4-METHYLTHIOBUTANOIC-ACID AND L

METHIONINE IN THE BROILER CHICK  
AUTHOR: KNIGHT C D; **DIBNER J J**  
AUTHOR ADDRESS: MONSANTO COMPANY, 700 CHESTERFIELD VILLAGE PARKWAY, ST.  
LOUIS, MO. 63198.  
JOURNAL: J NUTR 114 (11). 1984. 2179-2186. 1984  
FULL JOURNAL NAME: Journal of Nutrition  
CODEN: JONUA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: The absorption of [1-14C]2-hydroxy-4-(methylthio)butanoic acid (HMB) and L-methionine (L-Met) was evaluated in the broiler chick. Methods included in vitro intestinal tissue uptake and appearance in the plasma from ligated intestinal segments in situ, and from crop intubations. Intestinal uptake of L-Met was partially inhibited with 2,4-dinitrophenol, while HMB uptake was unaffected. The uptake of L-Met conformed to Michaelis-Menten kinetics except that no plateau was reached. This indicated that L-Met absorption may be accomplished by both concentration and energy-dependent processes. The in vitro uptake of HMB in relation to concentration was linear indicating that its absorption was concentration dependent. Absorption of HMB and L-Met after crop intubation indicated higher plasma concentrations of HMB than of L-Met, particularly at higher doses. Both compounds were absorbed at similar rates at concentrations 50-100 times below physiological concentrations and absorption rates were different at different small intestinal locations. These results indicate that the ability to use HMB as a source of methionine would not be limited by absorption.

13/7/47 (Item 47 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04602484 BIOSIS NO.: 000079015521  
CONVERSION OF 2-HYDROXY-4-METHYLTHIOBUTANOIC-ACID TO L-METHIONINE IN THE  
CHICK: A STEREOSPECIFIC PATHWAY  
AUTHOR: **DIBNER J J**; KNIGHT C D  
AUTHOR ADDRESS: MONSANTO COMPANY, 800 N. LINDBERGH BLVD., ST. LOUIS, MO.  
63167.  
JOURNAL: J NUTR 114 (9). 1984. 1716-1723. 1984  
FULL JOURNAL NAME: Journal of Nutrition  
CODEN: JONUA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: Conversion of 2-hydroxy-4-(methylthio)butanoic acid (HMB) to L-methionine (L-Met) was studied by using chick liver homogenates. The 1st step was found to be stereospecific with different enzymes for the D- and L-isomers of HMB. L-HMB was the substrate for L-2-hydroxy acid oxidase, a peroxide-producing flavoenzyme found in peroxisomes of liver and kidney. The enzyme for D-HMB, identified as mitochondrial D-2-hydroxy acid dehydrogenase, had not been previously described in the chick. This enzyme was found in every tissue tested including intestinal mucosa and skeletal muscle. Thus, D-HMB could be used by any organ for protein synthesis, like L-Met itself. These results provide a biochemical explanation of equimolar incorporation of HMB and DL-methionine (DL-Met) into chick hepatocyte protein in that the 2 HMB enzymes can simultaneously convert both HMB isomers to L-Met while only 1 enzyme, D-2-amino acid oxidase, converts D-Met to L-Met.

13/7/48 (Item 48 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04560352 BIOSIS NO.: 000029083389

AGE-DEPENDENT CHANGES IN THE METABOLISM OF D L-2

HYDROXY-4-METHYLTHIOBUTANOIC-ACID AND D METHIONINE

AUTHOR: **DIBNER J J**; KNIGHT C D

AUTHOR ADDRESS: MOSANTO CO., CHESTERFIELD, MO 63198, USA.

JOURNAL: 6TH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY,

ATLANTA, GA., USA, JAN. 22-23, 1985. POULT SCI 64 (SUPPL. 1). 1985. 14-15.

1985

CODEN: POSCA

DOCUMENT TYPE: Meeting

RECORD TYPE: Citation

LANGUAGE: ENGLISH

13/7/49 (Item 49 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

04300032 BIOSIS NO.: 000078029575

UTILIZATION OF SUPPLEMENTAL METHIONINE SOURCES BY PRIMARY CULTURES OF CHICK  
HEPATOCYTES

AUTHOR: **DIBNER J J**

AUTHOR ADDRESS: MONSANTO CO., 800 N. LINDBERGH BLVD., ST. LOUIS, MO. 63167.

JOURNAL: J NUTR 113 (10). 1983. 2116-2123. 1983

FULL JOURNAL NAME: Journal of Nutrition

CODEN: JCNUA

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: Utilization of 2-hydroxy-4-(methylthio) butanoic acid (HMB) as a substrate for protein synthesis was studied by using primary cultures of chick liver cells. Cultures were prepared by enzymatic dissociation of livers from week old Hubbard broiler chicks and were maintained for 4 days under nonproliferative conditions. Hepatocyte differentiation was verified by using dexamethasone induction of tyrosine aminotransferase activity. Conversion of [14C]HMB to L-methionine was shown by chromatographic analysis of hepatocyte protein hydrolysate and incorporation into protein was proven by cycloheximide inhibition of synthesis. When incorporation of HMB was compared to that of D-methionine (DLM) equimolar quantities of the 2 sources were found in liver cell protein. At a cellular level, HMB and DLM are biochemically equivalent sources of methionine for protein synthesis.

13/7/50 (Item 50 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

(c) 2002 BIOSIS. All rts. reserv.

04194833 BIOSIS NO.: 000077020877

DEVELOPMENT OF AN IN-VITRO CLONOGENIC ASSAY FOR THE R-3327 RAT PROSTATIC  
ADENO CARCINOMA PERMITS COMPARISON OF THE PROLIFERATIVE POTENTIAL OF THE  
R-3327 R-3327A AND R-3327AT TUMORS

AUTHOR: **DIBNER J J**; NAKKEFF A

AUTHOR ADDRESS: SECTION CANCER BIOL., MALLINCKRODT INST. RADIOL.,

WASHINGTON UNIV. SCH. MED., 4511 FOREST PARK BLVD., ST. LOUIS, MO. 63108.

JOURNAL: PROSTATE 4 (3). 1983. 289-306. 1983

FULL JOURNAL NAME: Prostate

CODEN: PRSTD

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: The R3327 class of rat prostate tumors consists of both the androgen-dependent R3327 adenocarcinoma and androgen-independent sublines, the R3327At spindle cell tumor, and the R3327A, a squamous cell carcinoma. In vitro clonogenic cell assays were developed to measure and

compare systematically the proliferative potential of these tumors following various monodispersion techniques. Linear relationships between the number of monodispersed tumor cells cultured at low cell density and the number of colonies formed 10 days later establish these assays as the 1st quantitative cellular approach to those proliferative subpopulations ultimately responsible for the growth of these tumors. The name colony forming cell-prostatic adenocarcinoma (CFC-PA) was chosen to refer to the members of the proliferative subpopulation of the R3327 tumor. Ultrastructural comparison of R3327 adenocarcinoma tissue sections with the cells produced during culture provides evidence that the cells of the proliferative subpopulation may be derived from the acinar epithelium of the tumor.

13/7/51 (Item 51 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04151557 BIOSIS NO.: 000027061109  
THE METHIONINE ACTIVITY OF 2 HYXROXY-4-METHYL THIO BUTANOIC-ACID FOR  
BROILERS FED CRYSTALLINE AMINO-ACID DIETS  
AUTHOR: KNIGHT C D; **DIBNER J J**  
AUTHOR ADDRESS: MONSANTO COMPANY, ST. LOUIS, MO. 63167.  
JOURNAL: 73RD ANNUAL MEETING OF THE POULTRY SCIENCE ASSOCIATION, INC. POULT  
SCI 63 (SUPPL. 1). 1984. 129. 1984  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/52 (Item 52 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04147592 BIOSIS NO.: 000027057144  
UTILIZATION OF 2 HYDROXY-4-METHYL THIO BUTANOIC-ACID AND D L METHIONINE BY  
CHICK LIVER CELLS IN PRIMARY CULTURE  
AUTHOR: **DIBNER J J**  
AUTHOR ADDRESS: MONSANTO CO., ST. LOUIS, MO 63167.  
JOURNAL: 5TH ANNUAL MEETING OF THE SOUTHERN POULTRY SCIENCE SOCIETY,  
ATLANTA, GA., USA, JAN. 17-18, 1984. POULT SCI 63 (SUPPL. 1). 1984. 15-16.  
1984  
CODEN: POSCA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/53 (Item 53 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

04003474 BIOSIS NO.: 000076089042  
R-3327 PROSTATE ADENO CARCINOMA CLONOGENIC CELLS EPITHELIAL PROPERTIES AND  
HORMONE RESPONSE  
AUTHOR: **DIBNER J J**; NAKOFF A  
AUTHOR ADDRESS: SECTION OF CANCER BIOL., DIV. OF RADIATION ONCOL.,  
MALLINCKRODT INST. OF RADIOL., WASHINGTON UNIV. SCH. OF MED., 4511 FOREST  
PARK BLVD., ST. LOUIS, MO. 63108.  
JOURNAL: J NATL CANCER INST 70 (6). 1983. 1057-1066. 1983  
FULL JOURNAL NAME: Journal of the National Cancer Institute  
CODEN: JNCIA  
RECORD TYPE: Abstract  
LANGUAGE: ENGLISH

ABSTRACT: Cell colonies derived from the clonogenic tumor cell [colony-forming cell, prostate adenocarcinoma (CFC-PA)] assayed in vitro from the R3327 rat prostate adenocarcinoma demonstrate prostate acid phosphatase activity when assayed histochemically and convert testosterone to stanolone. The number of CFC-PA/104 cells plated in steroid-free cutlures was increased following the addition of testosterone or stanolone and decreased following the addition of 17.beta.-estradiol. The decreased rate of growth of the R3327 tumor in castrated male inbred Copenhagen rats when compared to the growth measured in normal (intact) male and female inbred Copenhagen rats was reflected in a large decrease in the number of CFC-PA/104 cells plated from tumors grown in castrated male rats when compared to the values obtained from tumors that were grown in normal male and female rats. The replacement of fetal calf serum with normal male or castrate male rat serum resulted in little change in CFC-PA/104 cells plated in cultures established from tumors grown in castrated rats, although significant increases in CFC-PA were observed in cultures established from tumors grown in normal male or female rats.

13/7/54 (Item 54 from file: 5)  
DIALOG(R)File 5:Biosis Previews(R)  
(c) 2002 BIOSIS. All rts. reserv.

03084623 BIOSIS NO.: 000020027742  
IN-VITRO CLONOGENIC ASSAY FOR THE R-3327 RAT PROSTATE ADENO CARCINOMA  
AUTHOR: **DIBNER J J**; **NAKEFF A**  
AUTHOR ADDRESS: WASH. UNIV., ST. LOUIS, MO. 63110, USA.  
JOURNAL: 31ST ANNUAL MEETING OF THE TISSUE CULTURE ASSOCIATION, ST. LOUIS, MO., USA, JUNE 1-5, 1980. IN VITRO 16 (3). 1980. 212. 1980  
CODEN: ITCSA  
DOCUMENT TYPE: Meeting  
RECORD TYPE: Citation  
LANGUAGE: ENGLISH

13/7/55 (Item 1 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

08247054 Genuine Article#: 263BN Number of References: 37  
Title: Effect of turkey (*Meleagris gallopavo*) breeder hen age and egg size on poult development. 2. Intestinal villus growth, enterocyte migration and proliferation of the turkey poult  
Author(s): Applegate TJ; **Dibner JJ**; Kitchell ML; Uni Z; Lilburn MS  
(REPRINT)  
Corporate Source: OHIO STATE UNIV, OHIO AGR RES & DEV CTR, DEPT ANIM SCI/WOOSTER//OH/44691 (REPRINT); OHIO STATE UNIV, OHIO AGR RES & DEV CTR, DEPT ANIM SCI/WOOSTER//OH/44691; NOVUS INT INC, /ST CHARLES//MO/63304; HEBREW UNIV JERUSALEM, FAC AGR, DEPT ANIM SCI/IL-76100 REHOVOT//ISRAEL/  
Journal: COMPARATIVE BIOCHEMISTRY AND PHYSIOLOGY B-BIOCHEMISTRY & MOLECULAR BIOLOGY, 1999, V124, N4 (DEC), P381-389  
ISSN: 0305-0491 Publication date: 19991200  
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND  
Language: English Document Type: ARTICLE  
Abstract: Villus growth, enterocyte migration and proliferation were measured in the small intestine of poults (*Meleagris gallopavo*) to determine if hen age and/or egg size influences these characteristics during the first week after hatching. At hatching, distal jejunal villi were 22.8  $\mu$ m longer in poults from the older (48 weeks) versus the younger (34 weeks) hens ( $P < 0.05$ ). Similarly, labeled enterocytes in distal jejunal sections from poults from the older hens had migrated 28

mu m (10%) farther along the crypt-villus axis at hatching, as compared to poults from the younger hens ( $P < 0.05$ ). Villus growth differences and enterocyte migration were not consistently affected by hen age or egg weight class in poults from 1 to 7 days old. These results suggest that even though intestinal villi may be more advanced developmentally at hatch in poults from the older hens, however post-hatch growth of the intestine or the poult is not affected by hen age or egg weight class. (C) 1999 Elsevier Science Inc. All rights reserved.

13/7/56 (Item 2 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

07446772 Genuine Article#: 166RA Number of References: 51  
Title: Early feeding and development of the immune system in neonatal poultry  
Author(s): **Dibner JJ (REPRINT)** ; Knight CD; Kitchell ML; Atwell CA; Downs AC; Ivey FJ  
Corporate Source: NOVUS INT INC, 20 RES PK DR, MISSOURI RES PK/ST CHARLES//MO/63304 (REPRINT)  
Journal: JOURNAL OF APPLIED POULTRY RESEARCH, 1998, V7, N4 (WIN), P425-436  
ISSN: 1056-6171 Publication date: 19980100  
Publisher: APPLIED POULTRY SCIENCE INC, PO BOX 80286, ATHENS, GA 30608  
Language: English Document Type: ARTICLE  
Abstract: The objective of this research was to evaluate the effect of early feeding on the development of the immune system in broiler chicks. A hydrated nutritional supplement formulated specifically for the first 2 to 3 days of post-hatch life was provided to the chicks on the day of hatch and the 2 subsequent days. The nutritional content of this supplement was optimized to achieve maximum performance at 6 wk following ad libitum consumption of starter feed and water. Control hatchlings received neither feed nor water. After the initial treatment, all birds were fed the same corn-soy diet ad libitum. Immune development was assessed by measuring bursa weight, biliary immunoglobulin A (IgA) levels, appearance of germinal centers in the cecal tonsils, and resistance to an oral challenge with coccidial oocysts. Data indicate that provision of the optimum balance of nutrients immediately after hatch results in heavier bursa weights, earlier appearance of biliary IgA and germinal centers, and an improved resistance to disease challenge.

13/7/57 (Item 3 from file: 34)  
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci  
(c) 2002 Inst for Sci Info. All rts. reserv.

06594992 Genuine Article#: ZD408 Number of References: 17  
Title: The relative effectiveness of 2-hydroxy-4-(methylthio) butanoic acid and DL-methionine in young swine  
Author(s): Knight CD (REPRINT) ; Atwell CA; Wuelling CW; Ivey FJ; **Dibner JJ**  
Corporate Source: NOVUS INT INC, /ST CHARLES//MO/63304 (REPRINT)  
Journal: JOURNAL OF ANIMAL SCIENCE, 1998, V76, N3 (MAR), P781-787  
ISSN: 0021-8812 Publication date: 19980300  
Publisher: AMER SOC ANIMAL SCIENCE, 1111 NORTH DUNLAP AVE, SAVOY, IL 61874  
Language: English Document Type: ARTICLE  
Abstract: We compared the effectiveness of 2-hydroxy-4-(methylthio) butanoic acid (HMB) and DL-methionine (DLM) as sources of L-methionine activity in methionine-deficient primary cultures of pig liver cells and methionine-deficient early-weaned pigs. Viable hepatocytes were obtained from minced pig liver and maintained in a high density, differentiated, nonproliferation cell culture system. Culture medium was supplemented with HMB, DLM, or L-methionine, and cells were pulse-dosed with L[C-14(U)]leucine for 24 h to determine the level of



protein synthesis. Leucine incorporation per milligram of protein indicated a six-to eightfold increase in protein synthesis ( $P < .01$ ) with methionine levels between 5 and 10 CIM, regardless of source of methionine activity. Two 24-pen replicate methionine dose titrations were conducted with 95 early-weaned commercial crossbred pigs. The pelleted corn, dried whey, and porcine plasma basal diet contained 1.5% lysine, .23% methionine, and .48% cystine and was supplemented with 0, .05, or .10% methionine activity as DLM or HMB for 21 d. There was a 134, 104, and 61% increase ( $P < .01$ ) in cumulative ADG for each successive week on study with a 30 and 19% improvement in feed/gain ( $P < .01$ ) after 7 and 14 d. Performance responses due to source of methionine activity did not differ and slope ratio potency determinations (gain vs intake of methionine source) of HMB vs DLM indicated a 119, 111, and 95% relative activity for cumulative weekly performance. These results support the hypothesis that HMB and DLM provide equimolar levels of methionine activity in swine.

13/7/58 (Item 1 from file: 73)  
DIALOG(R) File 73:EMBASE  
(c) 2002 Elsevier Science B.V. All rts. reserv.

03023112 EMBASE No: 1985017078  
Proliferative properties of the clonogenic cells of the R3327 prostate adenocarcinoma  
**Dibner J.J.**; Nakeff A.  
Section of Cancer Biology, Mallinckrodt Institute of Radiology,  
Washington University School of Medicine, St. Louis, MO 63108 United States  
In Vitro ( IN VITRO ) (United States) 1983, 19/3 I (179-190)  
CODEN: ITCSA  
DOCUMENT TYPE: Journal  
LANGUAGE: ENGLISH

13/7/59 (Item 1 from file: 155)  
DIALOG(R) File 155:MEDLINE(R)

13032252 21589293 PMID: 11732679  
Uptake of DL-2-hydroxy-4-methylthio-butanoic acid (DL-HMB) in the broiler liver in vivo.  
Wang S; Bottje W G; Song Z; Beers K; Vazques-Anon M; **Dibner J J**  
Department of Poultry Science, University of Arkansas, Fayetteville 72701, USA.  
Poultry science (United States) Nov 2001, 80 (11) p1619-24, ISSN 0032-5791 Journal Code: 0401150  
Document type: Journal Article  
Languages: ENGLISH  
Main Citation Owner: NLM  
Record type: Completed  
The methionine source DL-2-hydroxy-4methylthio-butanoic acid (DL-HMB; Alimet feed supplement) is widely used in the poultry industry. The purpose of this study was to determine the capacity of the broiler liver to remove DL-HMB from the circulation. Cannulae were implanted in the carotid artery and hepatic and hepatic portal veins in anesthetized male broilers (3.33 +/- 0.13 kg BW). In Experiment 1, birds (n = 5) were infused with DL-HMB solutions (diluted in saline, pH 7.2 to 7.4) into the hepatic portal vein at rates ranging from 4.4 to 22 mg/min per kg BW, whereas in Experiment 2, birds (n = 6) were infused with DL-HMB at rates ranging from 2.2 to 4.4 mg/min per kg BW. Plasma samples from each vessel were obtained before and after each 10-min DL-HMB infusion period with a 10-min clearance period allowed between each DL-HMB infusion. Regression analysis revealed a highly significant correlation in the amount of DL-HMB entering the liver via afferent vessels (afferent DL-HMB) and DL-HMB removed by the liver ( $y = 0.86(x) - 173$ ,  $r^2 = 0.98$ ). The slope of this regression indicates that 86%

of DL-HMB entering in afferent blood (i.e. from both the hepatic artery and hepatic portal vein) was removed or that the liver apparently metabolized 86% of the DL-HMB that entered the liver. The results indicate that the broiler liver has the capacity to remove DL-HMB from the circulation far in excess of that needed to metabolize DL-HMB that would enter the liver following gastrointestinal absorption in birds fed a conventional poultry diet. In addition, present results implicate the liver as a major site of removal from circulation and further metabolism of DL-HMB in chickens.

Record Date Created: 20011204

13/7/60 (Item 2 from file: 155)  
DIALOG(R)File 155:MEDLINE(R)

05956231 89041845 PMID: 3186593

Absorption of 14C-2-hydroxy-4-(methylthio)butanoic acid (Alimet) from the hindgut of the broiler chick.

**Dibner J J**; Knight C D; Swick R A; Ivey F J

Monsanto Company, Animal Science Division, St. Louis, Missouri 63198.

Poultry science (UNITED STATES) Sep 1988, 67 (9) p1314-21, ISSN 0032-5791 Journal Code: 0401150

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The purpose of these studies was to examine the role of the hindgut of the broiler chick in the absorption of 2-hydroxy-4-(methylthio)butanoic acid (HMB). When 14C-HMB was delivered directly into the hindgut, the rate of absorption from this gastrointestinal site was about 40% of the administered dose per hour. Plasma radiolabel appearance indicated that the 14C-HMB lost from the hindgut was being absorbed into the bloodstream of the bird. Decarboxylation experiments using cecal microorganisms showed that the loss of 14C-HMB could not be accounted for by bacterial metabolism. When birds were dosed with radiolabelled HMB and tissue samples were tested, results showed that the 14C-HMB that was absorbed from the hindgut was incorporated into protein in a dose-related manner. In addition, an equimolar, equal specific activity intraperitoneal dose of HMB did not alter the rate of HMB adsorption from the hindgut. This indicates that HMB absorption from the gut is not limited by HMB already in the body tissues. This result confirmed that the rate of HMB diffusion into the blood and its conversion to methionine in body tissues were sufficient to maintain the concentration gradient required for the continued absorption of HMB. Finally, whole body autoradiography comparing 35S-HMB and 35S-DL-methionine showed no substantial differences in terms of label density or distribution. These studies demonstrate that 14C-HMB disappears from the lumen of the large intestine and ceca when it is administered directly into the hindgut. This research confirms that HMB is absorbed throughout the entire gastrointestinal system.

Record Date Created: 19881208

13/7/61 (Item 1 from file: 434)  
DIALOG(R)File 434:SciSearch(R) Cited Ref Sci  
(c) 1998 Inst for Sci Info. All rts. reserv.

05781090 Genuine Article#: SR343 Number of References: 1

Title: CORRECTION

Author(s): **DIBNER JJ**

Journal: JOURNAL OF NUTRITION, 1984, V114, N5, P989

Language: ENGLISH Document Type: CORRECTION, ADDITION

13/7/62 (Item 2 from file: 434)  
DIALOG(R)File 434:SciSearch(R) Cited Ref Sci  
(c) 1998 Inst for Sci Info. All rts. reserv.

05191237 Genuine Article#: QV322 Number of References: 26  
 Title: R3327 PROSTATE ADENOCARCINOMA CLONOGENIC CELLS - EPITHELIAL  
 PROPERTIES AND HORMONE RESPONSE .3.  
 Author(s): **DIBNER JJ**; NAKKEFF A  
 Corporate Source: WASHINGTON UNIV, SCH MED, MALLINCKRODT INST RADIOL, DIV  
 RADIAT ONCOL, CANC BIOL SECT/ST LOUIS//MO/63108  
 Journal: JOURNAL OF THE NATIONAL CANCER INSTITUTE, 1983, V70, N6, P  
 1057-1066  
 Language: ENGLISH Document Type: ARTICLE

13/7/63 (Item 3 from file: 434)  
 DIALOG(R) File 434: SciSearch(R) Cited Ref Sci  
 (c) 1998 Inst for Sci Info. All rts. reserv.

00297328 Genuine Article#: T8237 Number of References: 1  
 Title: EFFECTS OF PORCINE COMPLEX II ON AMINOACYLATION OF ASPARTIC-ACID AND  
 ISOLEUCINE  
 Author(s): **DIBNER JJ**  
 Corporate Source: STATE UNIV NEW YORK/BINGHAMTON//NY/13901  
 Journal: ABSTRACTS OF PAPERS OF THE AMERICAN CHEMICAL SOCIETY, 1974, P160  
 Language: ENGLISH Document Type: MEETING ABSTRACT  
 ? ds

Set	Items	Description
S1	1547	SPOROCTYST? AND (EIMERIA OR TENELLA OR NECATRIX OR ACERVULI- NA OR PARECOX OR BRUNETTI OR MITIS)
S2	715	RD S1 (unique items)
S3	14	S2 AND (OVO OR EGG?)
S4	18	S2 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S5	17	S4 NOT S3
S6	36420	(EIMERIA OR TENELLA OR NECATRIX OR ACERVULINA OR PARECOX OR BRUNETTI OR MITIS)
S7	984	S6 AND (OVO OR EGG?)
S8	120	S7 AND (IMMUNIZ? OR VACCIN? OR INJECT?)
S9	72	RD S8 (unique items)
S10	19	(TREAT? OR DISRUPT? OR GRIND?) AND S9
S11	53	S9 NOT (S3 OR S5 OR S10)
S12	137	AU='DIBNER J J' OR AU='DIBNER J.J.' OR AU='DIBNER JJ' OR A- U='DIBNER JULIA' OR AU='DIBNER JULIA J'
S13	63	RD S12 (unique items)
?		

---Logging off of Dialog---

? logoff

15jun02 15:36:30 User226352 Session D636.4  
 \$6.15 1.098 DialUnits File5  
 \$131.25 75 Type(s) in Format 7  
 \$131.25 75 Types  
 \$137.40 Estimated cost File5  
 \$0.83 0.140 DialUnits File6  
 \$0.83 Estimated cost File6  
 \$11.23 0.657 DialUnits File34  
 \$33.95 7 Type(s) in Format 7  
 \$8.40 2 Type(s) in Format 15  
 \$42.35 9 Types  
 \$53.58 Estimated cost File34  
 \$0.66 0.095 DialUnits File40  
 \$0.66 Estimated cost File40  
 \$0.51 0.106 DialUnits File41  
 \$0.51 Estimated cost File41  
 \$4.51 1.003 DialUnits File50

\$76.00 38 Type(s) in Format 7  
 \$76.00 38 Types  
 \$80.51 Estimated cost File50  
     \$0.49 0.130 DialUnits File65  
     \$0.49 Estimated cost File65  
     \$0.42 0.176 DialUnits File68  
     \$0.42 Estimated cost File68  
     \$3.25 0.452 DialUnits File71  
     \$3.25 Estimated cost File71  
     \$7.74 0.860 DialUnits File73  
     \$2.50 1 Type(s) in Format 7  
     \$2.50 1 Types  
 \$10.24 Estimated cost File73  
     \$2.03 0.398 DialUnits File76  
     \$7.40 4 Type(s) in Format 7  
     \$7.40 4 Types  
     \$9.43 Estimated cost File76  
     \$0.35 0.119 DialUnits File77  
     \$6.30 3 Type(s) in Format 7  
     \$6.30 3 Types  
     \$6.65 Estimated cost File77  
     \$1.01 0.290 DialUnits File94  
     \$1.35 1 Type(s) in Format 7  
     \$1.35 1 Types  
     \$2.36 Estimated cost File94  
     \$4.03 1.681 DialUnits File98  
     \$10.05 3 Type(s) in Format 7  
     \$10.05 3 Types  
 \$14.08 Estimated cost File98  
     \$1.55 0.304 DialUnits File103  
     \$1.55 Estimated cost File103  
     \$0.28 0.117 DialUnits File143  
     \$0.28 Estimated cost File143  
     \$1.70 0.486 DialUnits File144  
     \$1.70 Estimated cost File144  
     \$4.20 1.314 DialUnits File155  
     \$5.25 25 Type(s) in Format 7  
     \$5.25 25 Types  
     \$9.45 Estimated cost File155  
     \$0.85 0.313 DialUnits File156  
     \$0.85 Estimated cost File156  
     \$1.15 0.256 DialUnits File162  
     \$1.15 Estimated cost File162  
     \$1.04 0.115 DialUnits File172  
     \$1.04 Estimated cost File172  
     \$0.81 0.104 DialUnits File305  
     \$0.81 Estimated cost File305  
     \$0.31 0.088 DialUnits File369  
     \$0.31 Estimated cost File369  
     \$2.24 0.639 DialUnits File370  
     \$1.50 1 Type(s) in Format 7  
     \$1.50 1 Types  
     \$3.74 Estimated cost File370  
     \$7.57 0.603 DialUnits File399  
     \$8.25 3 Type(s) in Format 7  
     \$8.25 3 Types  
 \$15.82 Estimated cost File399  
     \$3.60 0.211 DialUnits File434  
     \$12.60 3 Type(s) in Format 14  
     \$12.60 3 Types  
 \$16.20 Estimated cost File434  
     OneSearch, 26 files, 11.755 DialUnits FileOS  
     \$3.68 TELNET  
 \$376.99 Estimated cost this search  
 \$493.20 Estimated total session cost 16.541 DialUnits

Logoff: level 02.05.06 D 15:36:30